A Dissertation on

PATTERN OF ACUTE KIDNEY INJURY AND ITS OUT COME SEEN IN MEDICALWARDS IN A TERTIARY CARE REFERRAL HOSPITAL,CHENNAI.

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032

In partial fulfilment of the Regulations
for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
CHENNAI – 600 001
APRIL 2016

CERTIFICATE BY INSTITUTION

This is to certify that **DR. S.JENNIE,** Post - Graduate Student (MAY 2013 TO APRIL 2016) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on

"**PATTERN OF ACUTE KIDNEY INJURY AND ITS OUT COME SEEN IN MEDICAL WARDS IN A TERTIARY CARE REFERRAL HOSPITAL, CHENNAI – 600001"** under my guidance and supervision in partial fulfilment of the regulations laid down by the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2016.

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DECLARATION

I **Dr. S.JENNIE** declare that I carried out this work on "**PATTERN OF ACUTE KIDNEY INJURY AND ITS OUT COME SEEN IN MEDICAL WARDS IN A TERTIARY CARE REFERRAL HOSPITAL, CHENNAI – 600001"** at the Medical Wards of Government Stanley Hospital during the period of January 2015 to september 2015. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Degree examination in General Medicine.

Dr. S.JENNIE

ACKNOWLEDGEMENT

At the outset I thank our Dean **Dr. CRISTIAN ISAAC MOSES,MD.,FICP.,FACP.,** for permitting me to carry out this study in our hospital.

I express my profound thanks to my esteemed Professor and Teacher **Dr. R. JAYANTHI, M.D.,** Professor and HOD of Medicine, Stanley Medical College Hospital, for encouraging and extending invaluable guidance to perform and complete this dissertation.

I also express my profound thanks to my esteemed Professor and Teacher **Dr.EDWIN FERNANDO, M.D,D.M,FRCP,** Head of the Department of Nephrology, Stanley Medical College, who was also my Guide for encouraging and extending invaluable guidance to perform and complete this dissertation

I wish to thank **Dr.NALINI KUMARAVELU, M.D.,** Professor of my unit, Department of Medicine, Stanley medical college Hospital for her valuable suggestions, encouragement and advice.

I wish to thank **DR. N. KARUNAKARAN MD.,** Assistant Professor, Department of Medicine, Stanley medical college Hospital for his valuable suggestions, encouragement and advice.

I express my sincere thanks to **Dr. G. SATHYA M.D, D.M,** Assistant professor, department of General Medicine for her support.

I thank **Dr. ARUN MURUGAN M.D,** Professor of Community Medicine, Kilpauk Medical College for his timely help in my statistical analysis.

I sincerely thank the members of Institutional Ethical Committee, Stanley Medical College for approving my dissertation topic.

I thank all my Colleagues, House Surgeons, and Staff nurses and other para-medical workers for their support.

I sincerely thank my parents for their extended support.

Last but not the least, I sincerely thank all those patients who participated in this study, for their kind cooperation.

Abstract

Introduction

We conducted an observational study to evaluate the trends of AKI and its outcome seen in medical wards in a tertiary care referral hospital.

Methods

The study was a prospective observational study. It was conducted at Stanley Medical College . Data were collected from Medical wards in Government Stanley Hospital. Totally 102 patients of AKI were included in this study from january 2015 to august 2015. The main trends of AKI presentation and its outcome were assessed..

Results

Of 102 patients admitted, 42 had a sepsis-related diagnosis (42.41%). Among septic patients 37(40.66%) were recovered from AKI ; 5 patients (45.45%) were not recovered (P<0.7523) ; 17 patients (17.16%) had cardiovascular disease related AKI; 12 patients (12.12%) had developed AKI due to drugs and poisons, of which 8 patients (8.79%) were recovered and 4 patients (36.36%) were not recovered (P <0.0030).

For AKI stratified by RIFLE (risk of renal failure, injury to the kidney, failure of kidney function, loss of kidney function and end-stage kidney disease) category, 43.96 % of patients belonged to the risk category, 30.77 % to the injury category. Of 34 patients in failure category 23 (25.27%) were recovered 11 (100%) were not recovered (P< 0.0388) .

In patients belonging to recovered group, the over all mean serum creatinine values were 2.05 mg/dl, in non recovered group it is 3.42 mg/dl(P value is < 0.0173).

In patients of recovered group, the overall mean urine output values is 783 ml/day; in deterioration group, 445 ml/day (P value is< 0.0048.)

CONCLUSIONS

1.Common causes of AKI in this study include, sepsis, cardiovascular diseases, drugs and poisons, and diarrhoeal disease in order of occurrence.

2. Among the patients who had AKI due to sepsis scrub typhus topped the list followed by leptospirosis and falciparum malaria.

3.Higher values of serum creatinine at admission and oliguria were the most significant factors that contributed to non recovery from acute kidney injury.

ABBREVIATIONS

AGN – Acute glomerulonephritis

AIN - Acute interstitial nephritis

AKI – Acute kidney injury

AKIN – Acute kidney injury network

ARF – Acute renal failure

ATN – Acute tubular necrosis

BPH – Benign prostatic hypertrophy

BUN – Blood urea nitrogen

CRRT - Continuous renal replacement therapy

CI-AKI – Contrast induced acute kidney injury

CKD – Chronic kidney disease

FeNa – Fractionated excretion of sodium

GFR – Glomerular filteration rate

HIT - Heparin-induced thrombocytopenia

HMW – High molecular weight

ICU – Intensive care unit

IGF – Insulin like growth factor

IHD - Intermittent hemodialysis

KDIGO – Kidney disease: Improving global outcomes

KIM - kidney injury molecule

LOH – Loop of henle

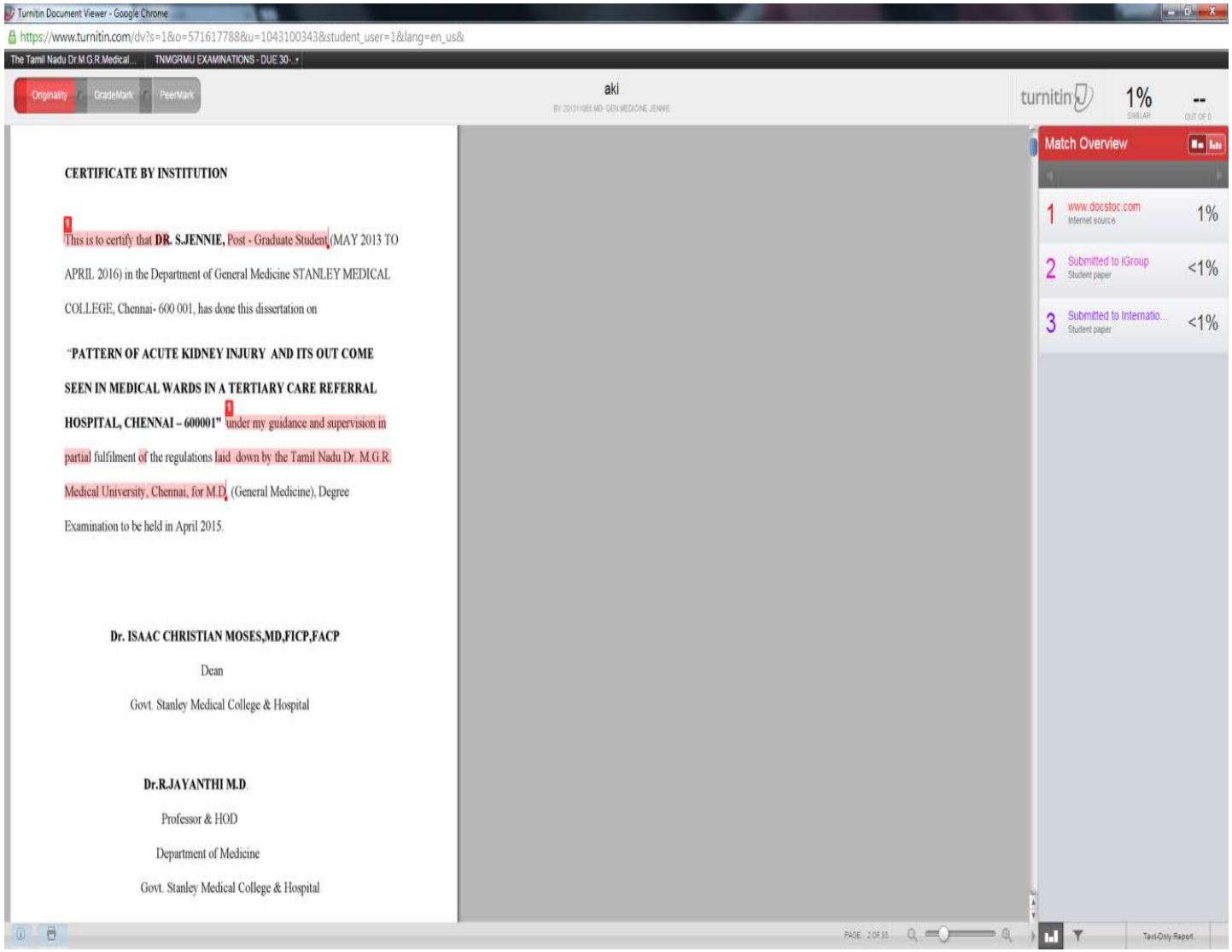
MPGN – Membranous proliferative glomerulo nephritis

NGAL - Neutrophil gelatinase associated lipocalin

PCT – Proximal convoluted tubules

RIFLE – Risk,Injury,Failure,Loss of kidney function,and End stage Renal disease

RRT - Renal replacement therapy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Introduction

INTRODUCTION

Acute kidney injury is the abrupt deterioration of function of kidney, leading to retention of urea and other nitrogenous waste products and with normal or decreased urine output or both and there is dysregulation of extracellular fluid volume and electrolytes.

AKI - The term has widely replaced the term acute renal failure (ARF), it reflects that, the understanding of smaller decrements in renal function will not result in overt organ failure are of important clinical relevance and are associated with increased mortality and morbidity. The term acute renal failure is now used for severe AKI, which usually indicates the need for renal replacement therapy.

The spectrum of acute kidney injury ranges from mild to severe, at times requiring renal replacement therapy. The preliminary workup includes a detailed history to know the systemic illnesses and use of nephrotoxic drugs that might cause decline in renal perfusion or the causes which directly derange renal function.

Meticulous clinical examination is very important to assess intravascular volume status and skin rashes which indicates systemic illness. The first laboratory evaluation includes measurement of serum creatinine, complete blood count, fractional excretion of sodium and urinalysis. Imaging of the

kidneys by ultrasonography should be performed in all patients, especially in elderly males, to rule out obstructive urinary pathology.

INITIAL MANAGEMENT OF ACUTE KIDNEY INJURY

(i)fluid resuscitation,

(ii)avoidance of contrast media exposure

(iii)avoidance of nephrotoxic medications

(iv) correction of electrolyte imbalances.

INDICATIONS OF RENAL REPLACEMENT THERAPY

(i)Refractory hyperkalemia

(ii)Volume overload status

(iii)Pericarditis

(iv)Uremic encephalopathy,

(v)Intractable acidosis

(vi)Pleuritis,

Recognition of risk factors like cardiac surgery, hypovolemia, shock, sepsis, diabetes mellitus, older age, pre existing chronic kidney disease, liver failure, cardiac failure and infusion of contrast agents are important.

Team-based approaches are appreciated to prevent, to diagnose early, and manage the patient aggressively to bring out good outcomes.

Review of Literature

REVIEW OF LITERATURE

MOLECULAR AND CELLULAR BIOLOGY OF KIDNEY

Most highly differentiated organs in the body is kidney. Almost 30 different cell types forms a multiple number of capillaries which filters and segmented nephrons, which was enveloped by a interstitium. This cellular diversity is a reason for variety of complex physiologic processes. Regulation of blood pressure, Endocrine functions, solute and water transport, intraglomerular hemodynamics, acid-base balance, and removal of drug metabolites are all accomplished by complex mechanisms kidney.

Glomerular Filtration Regulation

Approximately 20% of the cardiac output (ie.,1 litre/min) enters to renal blood flow. Blood reaches via afferent arteriole leading into a glomerulus, there maximum amounts of fluids and solutes are filtered which forms tubular fluid. Coalesced glomerular capillaries at its distal end forms an efferent arteriole. Peritubular capillaries of cortex or vasa recta of medulla surrounding the tubules **.** Efferent arterioles regulate the hydrostatic pressure of capillary beds. Nephrons has 2 capillary beds; capillary beds drains into small venous branches; small venous branches coalesce & empty into larger veins; all larger veins ultimately forms Renal Vein.

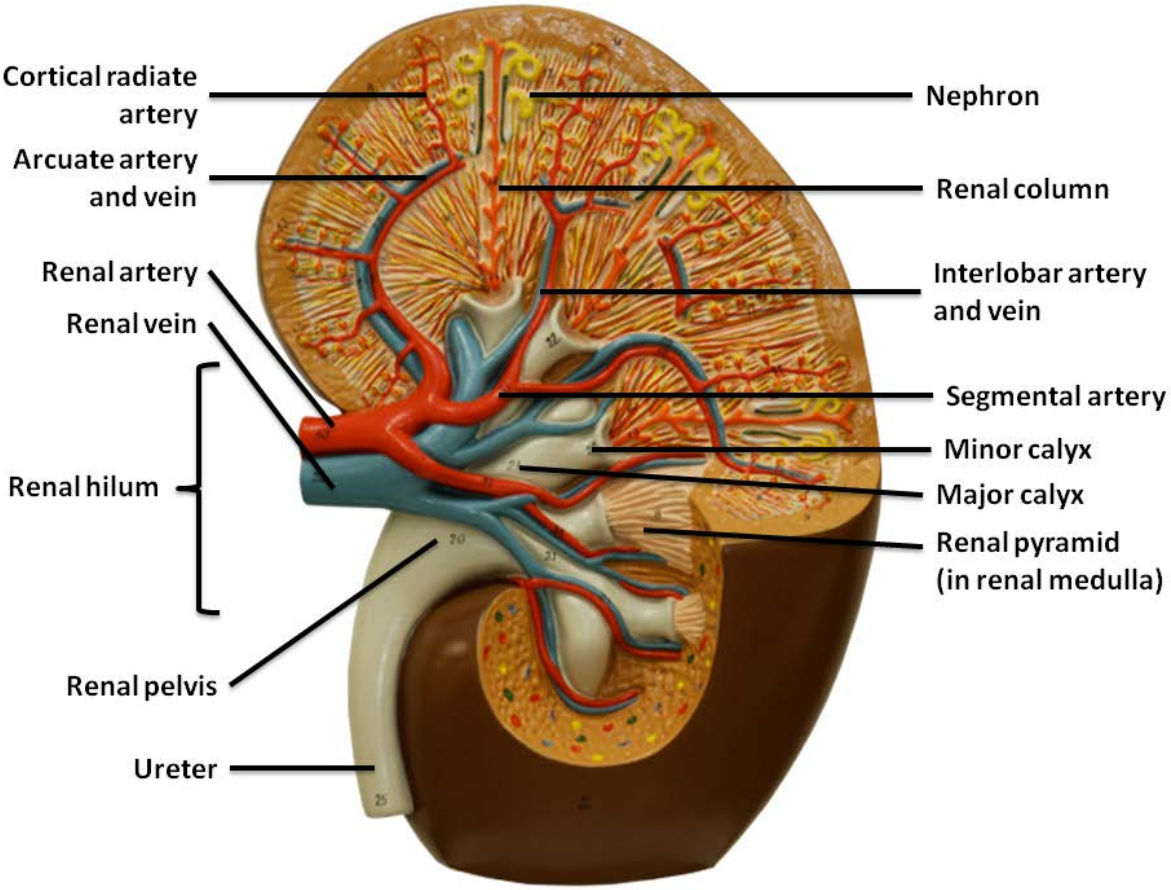
The primary driving force for glomerular filtration is depends upon the hydrostatic pressure gradient in the glomerular capillary. The concentration of

unfiltered plasma proteins determines the oncotic pressure of the capillary lumen, which partially reduces the hydrostatic pressure gradient and inhibits filtration. If the oncotic pressure increases in the glomerular capillary, the driving force for filtration comes down to zero while reaching the efferent arteriole.

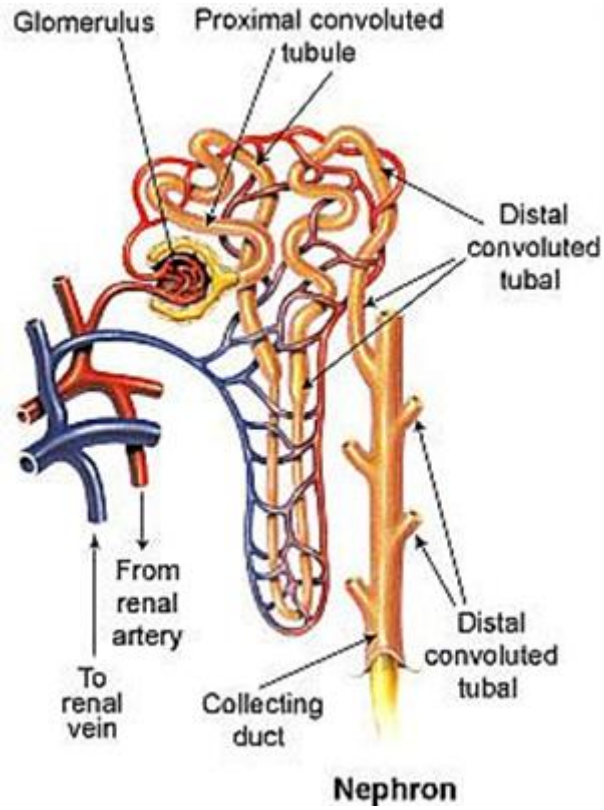
Three major factors that modulates the tone of the afferent & efferent arteriole results in autoregulation of glomerular filtration. Which includes (i)Autonomous vasoreactive (myogenic) reflex in the afferent arteriole, (ii)Tubuloglomerular feedback, and (iii)Angiotensin II–mediated efferent arteriolar vasoconstriction.

Tubular Transport Mechanism of kidney

The monolayers of cells lining the tubular segments ; the cells connected to one another by tight junction. Tight junctions separates the lumen from the interstitial spaces by forming an occlusive barriers. The interstitium facing basolateral membrane. Tubular lumen facing the apical membrane. This membrane protein & lipids allocated asymmetrically. Owing to this Tubular epithelial cells are polarised. The asymmetric assignment of membrane proteins, mediating transport processes.



Anatomy and microvasculature of kidney.



Epithelial Solute Transport

There are 2 types of epithelial transport.

(i)Cellular transport:

Movement of solutes & fluids across apical membrane & basolateral membrane via Transporters,Channels& Pumps.

(ii)Para cellular transport:

Movement of solutes & fluids via narrow passage between adjacent cells, ie.. nothing but Tight junctions. It has both leaky epithelia& tight epithelia. These are called low to high resistance epithelia because the flow of ion inside the

paracellular pathway forms the electrical resistance of cellular monolayer .The leaky epithelia located at proximal tubules & it is suitable for bulk fluid reabsorption , tight epithelia located at collecting ducts & it is suitable for refined control &regulation of transport .

Membrane Transport

The movement of solutes & water across membrane is made possible by channels, pumps& transporter.

(i)Active transport – Pumps

Ions moves against the chemical gradient. Pumps are electrogenic, means it creates an asymmetric electrostatic charges distributed across the membrane and create membrane potential examples;

(a)Na^+/K^+-ATPase ,

(b)H^+-ATPases,

(c) Ca^{2+}-ATPases.

(ii)Passive transport – Channels

The movements of solutes through membrane by simple diffusion. Examples

(a)Water channels (aquaporins),

(b)K^+ channels,

(c)Epithelial Na^+ channels

(d)Cl^- channels

(iii)Facilitated diffusion – Transporters

Facilitated by carriers or uniporters. Eg: GLUT2

Segmental Nephron Functions

PROXIMAL TUBULE:

Reabsorption of 60% of NaCl and H2O, ~90% of filtered HCo3 and most important nutrients such as glucose and amino acids occur at the proximal tubule. The PCT utilizes both cellular and paracellular transport mechanisms. The apical membrane of PCT has dense microvilli for extensive reabsorption called Brush border. Many leaky tight junctions present in PCT.

Solute and water passing via these tight junctions to enter the intercellular space peritubular capillaries absorb those solutes & water. Bulk fluid reabsorption by the proximal tubule is driven by high oncotic pressure and low hydrostatic pressure within the peritubular capillaries. Change in efferent arteriolar tone cause physiologic adjustments in GFR made by proportional changes in reabsorption called as glomerulo tubular balance.

Most solutes cellular transport at the proximal tubule is coupled to the Na^+ concentration gradient. Which is established by the activity of a basolateral Na^+/K^+-ATPase . Apart from the paracellular route, water reabsorption occurs

also through the cellular pathway; it is enabled by constitutively active water channels -aquaporin-1 present on basolateral and apical membranes.

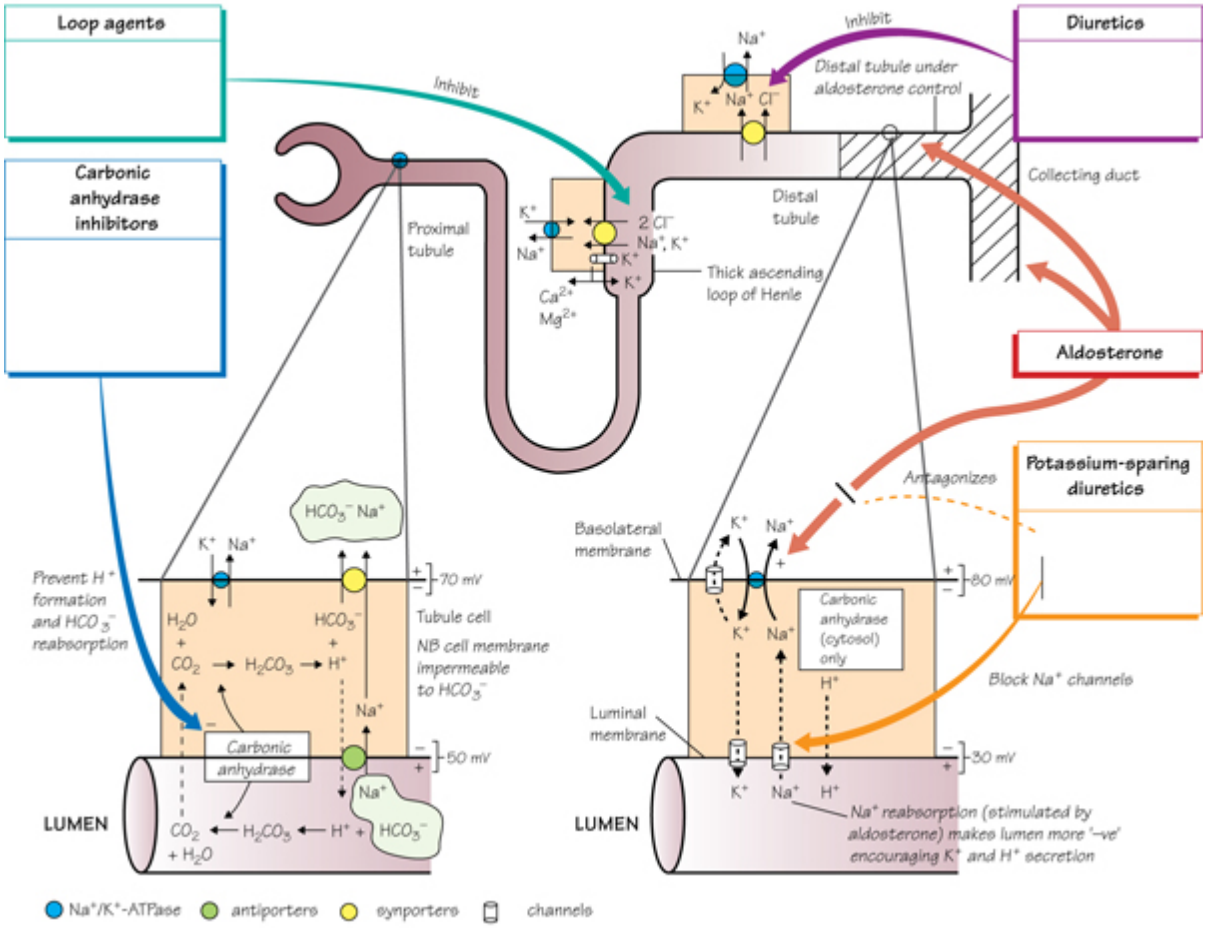
Bicarbonate reclaimed by a mechanism dependent on carbonic anhydrases at proximal tubular cells . Bicarbonate which is filtered ; titrated by protons delivered to the lumen by Na^+/H^+ exchange. The brush border carbonic anhydrase metabolises the resulting carbonic acid to water and carbon dioxide. Dissolved carbon dioxide then enter into the cell, there it is enzymatically hydrated by carbonic anhydrase to form carbonic acid again.

At last, intracellular carbonic acid disintegrates into free bicarbonate anions, protons , and bicarbonate exited from the cell through a basolateral Na^+/HCO_3^- co transporter. The above process is saturable, landed up in urinary bicarbonate excretion when plasma levels exceeds the physiologically normal range from 24 to 26 meq/L. Acetazolamide a carbonic anhydrase inhibitor is a class of weak diuretic agents, block reabsorption of bicarbonate at proximal tubule and are very useful for alkalinizing the urine.

Glucose reabsorption is nearly completed by the end of the proximal tubule. Glucose transport at cellular level is mediated by apical Na^+-glucose cotransport coupled with facilitated diffusion at basolateral by a glucose transporter. The above said process is also saturable, which leads to glycosuria when plasma levels exceed 180–200 mg/dL, as in diabetes mellitus.

The proximal tubule is having specific transporters capable of secreting a variety of bases - primary amine cations and organic acids - carboxylate anions. Organic anions transported by these systems are urate, and several protein-bound drugs not filtered at the glomerulus (cephalosporins, penicillins, and salicylates) and ketoacid anions,. Reabsorption of aminoacids at the proximal tubule, through distinct classes of Na^+-independent and Na^+-dependent transport systems,.

These transporters are specific for various groups of amino acids. Cystine, arginine, ornithine and lysine are absorbed by a system which has two proteins encoded by the SLC3A1 and SLC7A9genes. Mutations in above genes impair reabsorption of these amino acids and lead to cystinuria. Peptide hormones such as insulin, B_2-microglobulin, albumin and growth hormone, are absorbed by the proximal tubule through a process of absorptive endocytosis and are disintegrated in acidified endocytic lysosomes.



LOOP OF HENLE

There are three major segments:

(i)Descending thin limb,

(ii)Ascending thin limb, and

(iii)Ascending thick limb.

Approximately 20–25% of NaCl is reabsorbed in the loop of Henle, mainly at thick ascending limb. The LOH has an very important role in urinary concentration. It contributes to the process called counter current multiplication by generating hypertonic medullary interstitium. The LOH is the site for the most potent class of diuretic agents (loop diuretics) to act and contributes to reabsorption of magnesium and calcium ions.

The dense expression of active aquaporin-1 water channels over descending thin limb is highly water permeable. But water permeability is negligible in the ascending limb of Henle. There is secondary active salt transport by the $Na^+/K^+/2Cl^-$ co transporter (is the primary target for loop diuretics) on the apical membrane with basolateral Cl^- channels and Na^+/K^+-ATPase . K^+ is the limiting substrate for this co transporter . Tubular concentration of K^+ is parallel to plasma (about 4 meq/L), but transporter activity is balanced by K^+ recycling via an apical potassium channel.

Potassium recycling contributes to a positive electrostatic charge in the lumen compared to the interstitium which promotes divalent cation Magnesium and Calcium reabsorption via paracellular pathway. Loss-of-function mutations in CaSR leads to familial hypercalcemic hypocalciuria due to a blunted response of the thick ascending limb to extracellular Ca^{2+}.

Urine-concentrating ability is contributed by Loop of Henle by establishing a hypertonic medullary interstitium that promotes water reabsorption by the inner medullary collecting duct. Hypertonic medullary interstitium produced by a

counter current multiplication system using two counter current systems: (i) The loop of Henle -opposing ascending and descending limbs, and (ii)The vasa recta -medullary peritubular capillaries enveloping the loop.

The counter current flow in these two systems is the reason to maintain the hyper tonicity of the inner medulla, nevertheless NaCl absorption by the thick ascending limb is the primary initiating event. NaCl reabsorption without water dilutes tubular fluid and gives new osmoles to medullary interstitial fluid. Since the descending thin limb is highly water permeable, osmotic equilibrium occurs between the interstitial space and the descending limb tubular fluid , leading to increased solute trapping in the inner medulla. Maximum medullary interstitial osmolality also needs partial recycling of urea from the collecting duct.

DISTAL CONVOLUTED TUBULE

The DCT reabsorbs ~5% of the filtered NaCl with little water permeability. NaCl-transporting pathway uses an apical membrane, thiazide-sensitive Na^+/Cl^- co transporter in tandem with basolateral Na^+/K^+-ATPase and Cl^- channel . Ca^{2+}-selective channels in apical membrane and Na^+/Ca^{2+} exchange

in basolateral membrane maintain calcium reabsorption in distal convoluted tubule. Ca^{2+} reabsorption is stimulated by parathyroid hormone and it is inversely proportional to Na^{+} reabsorption. Familial hypertension with hyperkalemia are the features of pseudohypoaldosteronism type II or Gordon's syndrome.. Hyperkalemia may occur due to reduced activity of apical K^{+} channels in the collecting duct, a main route for K^{+} secretion.

COLLECTING DUCTS

The final composition of urine is modulated by collecting ducts. The two major divisions,

(i) Cortical collecting duct

(ii)Inner medullary collecting duct

contribution to reabsorbtion of ~4–5% of filtered Na^{+} .CT is important for hormonal regulation of salt and water balance. The cortical collecting duct has two cell types

(i)Principal cells - Na^{+}, water reabsorbing, and K^{+}-secreting cells. At this site aldosterone, mineralocorticoid receptor antagonists ,ie .,spironolactone, and K^{+}-sparing diuretics acts upon.

(ii)Type A and B intercalated cells

(a)Type A intercalated cells mediates bicarbonate reabsorption and acid secretion

(b)Type B intercalated cells mediates bicarbonate secretion and acid reabsorption.

All transport is being mediated by the cellular pathway for both principal cells and intercalated cells. In principal cells, apical Na^+ entry occurs through the epithelial Na^+ channel with basolateral exit through the Na^+/K^+-ATPase . This aldosterone tightly regulate Na^+ reabsorptive process.

Principal cells secrete K^+ through an apical membrane K^+- channel. Several factors govern the secretion of K^+. Importantly, the high intracellular potassium concentration created by Na^+/K^+-ATPase generates a favourable concentration gradient for K^+ secretion into tubular fluid. Reabsorption of Na^+ without any anion accompanied, the tubule becomes negative ,creating a electrical gradient for potassium secretion.

The driving force for K^+ secretion is blunted if Na^+ reabsorption is blocked, causes lack of excess urinary K^+ loss while treatment with potassium-sparing diuretics. K^+ secretion is also stimulated by aldosterone action.

Intercalated cells perform two types of transport: active H^+ transport mediated by H^+-ATPase (proton pump), and Cl^-/HCO_3^- exchange. Type A intercalated cells has an apical proton pump governs acid secretion and a basolateral Cl^-/HCO_3^- anion exchanger causes bicarbonate reabsorption .

But Type B intercalated cells has the anion exchanger on apical membrane to governs bicarbonate secretion, the proton pump of the basolateral membrane to

mediates acid reabsorption.

Many similarities between inner medullary collecting duct cells and principal cells of cortical collecting duct. Inner medullary collecting duct cells have vasopressin-regulated water channels - aquaporin-3 and -4 on the basolateral membrane and aquaporin-2 on the apical membrane,. The antidiuretic hormone vasopressin binds to the V2 receptor which triggers an intracellular signaling cascade.

This signalling cascade governs the insertion of water channels at inner medullary collecting duct cells apical membrane to stimulate increased water permeability. This increased permeability enables water reabsorption and end up in concentrated urine. Inner medullary collecting duct cells are water impermeable in the absence of vasopressin, and urine becomes diluted.

Acute kidney injury

Acute kidney injury is the abrupt deterioration of function of kidney, leads in retention of urea and other nitrogenous waste products or decreased urine output or both and there is dysregulation of extracellular fluid volume and electrolytes.

AKI - The term has widely replaced the term acute renal failure (ARF), it reflects that, the understanding of smaller decrements in renal function will not result in overt organ failure are of important clinical relevance and are associated with increased mortality and morbidity. The term acute renal failure is now used for severe AKI, usually indicates the need for renal replacement therapy.

AKI is mainly detected by measurement of serum creatinine. Creatinine is used for estimation of the glomerular filtration rate (GFR).

Using serum creatinine to quantitatively define AKI are associated with three problems :

(i)Serum creatinine will not precisely reflect the GFR of a patient in whom the GFR is not in steady state.

(a) In early stages of AKI, the sr. creatinine would be low, though the actual GFR is grossly reduced, because there may not have been enough time for sr. creatinine to accumulate .

(b)If the sr. creatinine is increasing, estimates of GFR depends on creatinine values will overestimate the real GFR

(c)Estimation of GFR will underestimate the actual GFR during recovery of renal function, when the sr. creatinine concentration is reducing.

(ii) During dialysis the creatinine is removed. As a result, once dialysis is initiated it is commonly not possible to assess renal function by measuring the serum creatinine. But the exception is if the serum creatinine continues to fall on days where hemodialysis is not performed, denotes the recovery of kidney function.

(iii)Multiple epidemiologic analysis and clinical trials have used various cut-off values for sr. creatinine to quantitatively define AKI

AKI is frequent clinical problem at intensive care unit: where it is associated with high mortality.[1] AKI is not a single entity but, ,a designation for a various group of conditions that have common diagnostic features.

STAGES OF AKI

Acute kidney injury is defined as an abrupt decline in kidney function within 48 hrs based on serum creatinine level elevation, a reduction in urine output, the requirement for dialysis (renal replacement therapy) or a combination of above factors. AKI is classified in three stages.

KDIGO GUIDELINES FOR AKI

Stage	Serum creatinine	Urine output
1	1.5-1.9×baseline or ≥0.3 mg/dl (≥26.5 mmol/l) increase	<0.5 ml/kg/h for 6-12 h
2	2.0-2.9×baseline	<0.5 ml/kg/h for >12 h
3	3.0×baseline, or increase in serum creatinine ≥4.0 mg/dl (≥353.6 mmol/l), or initiation of RRT, or decrease in eGFR <35 ml/min/1.73 m² for patients <18 years	<0.3 ml/kg/h for ≥24 h or anuria for ≥12 h

KDIGO: Kidney disease: Improving global outcomes; RRT: Renal replacement therapy; eGFR: Estimated glomerular filtration rate

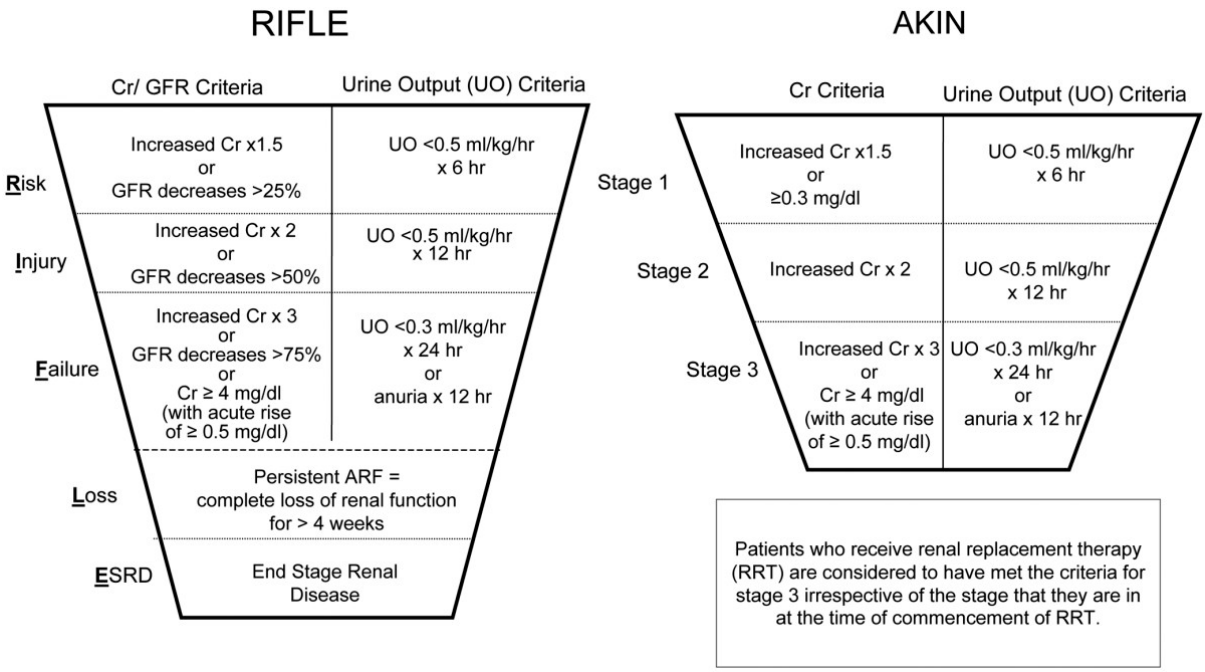
AKI is defined as any of the following :

Increase in Serum Creatinine by ≥0.3 mg/dl within 48 hours; or

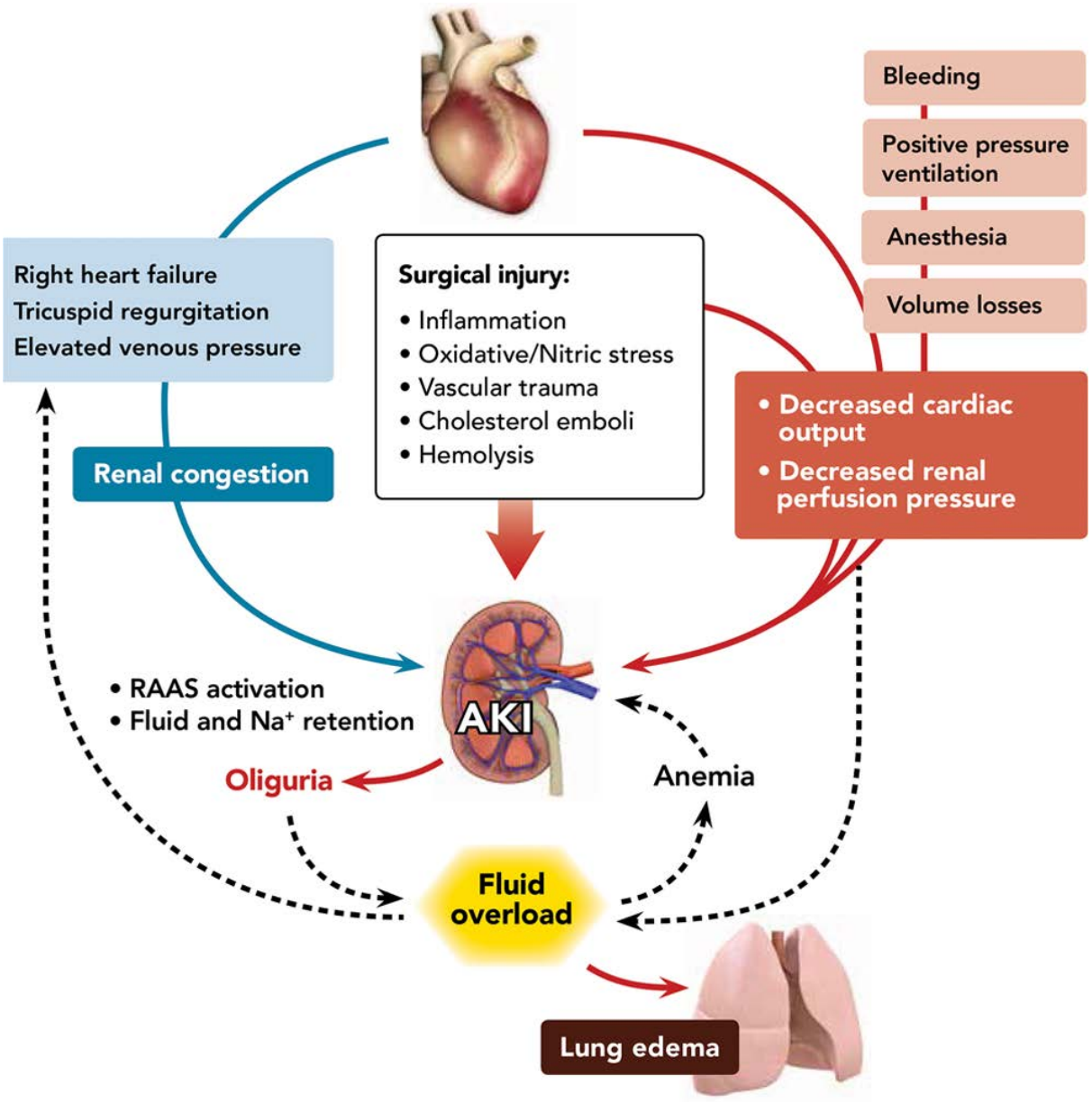
Increase in Serum Creatinine to ≥1.5 times of baseline, which is presumed to have occurred within the prior (1week)7 days; or

Urine volume < 0.5 ml/kg/h for 6 hours.

AKI is categorised into 5 stages by RIFLE criteria.



AKIN criteria is identical to RIFLE criteria in first 3 stages. Exception with shorter timeframe of less than 48 hrs and lower creatinine threshold o >0.3mg/dl from baseline value to peak value.



Etiopathogenesis of AKI.

EPIDEMIOLOGY

In acute care hospital admission AKI affects 5 to 7 % of patients , up to 30% of admissions in the critical care unit. In the developing world along with many other disease AKI is also a major medical complication, especially in the setting of diarrheal illnesses, infectious diseases like leptospirosis and Malaria . The incidence of AKI has increased by more than fourfold in the US since 1988 and is been calculated to have annual incidence of 500 per 1 lakh population[2], higher than annual incidence of stroke. AKI is associated with a remarkably increased risk of death in inpatients, particularly in patients admitted to the critical care unit where the hospital mortality percentage may exceed 50%.

PREVALENCE OF AKI IN DEVELOPING WORLD

The epidemiology of AKI differs enormously between developing and developed countries, because of differences in geography, economics, demographics, and comorbid disease burden. Most of the etiologies for AKI are geographic-specific such as envenomations from spiders ,snakes, bees and caterpillars ; infectious etiologies such as leptospirosis and malaria and rhabdomyolysis due to crush injuries resultant from earthquakes.

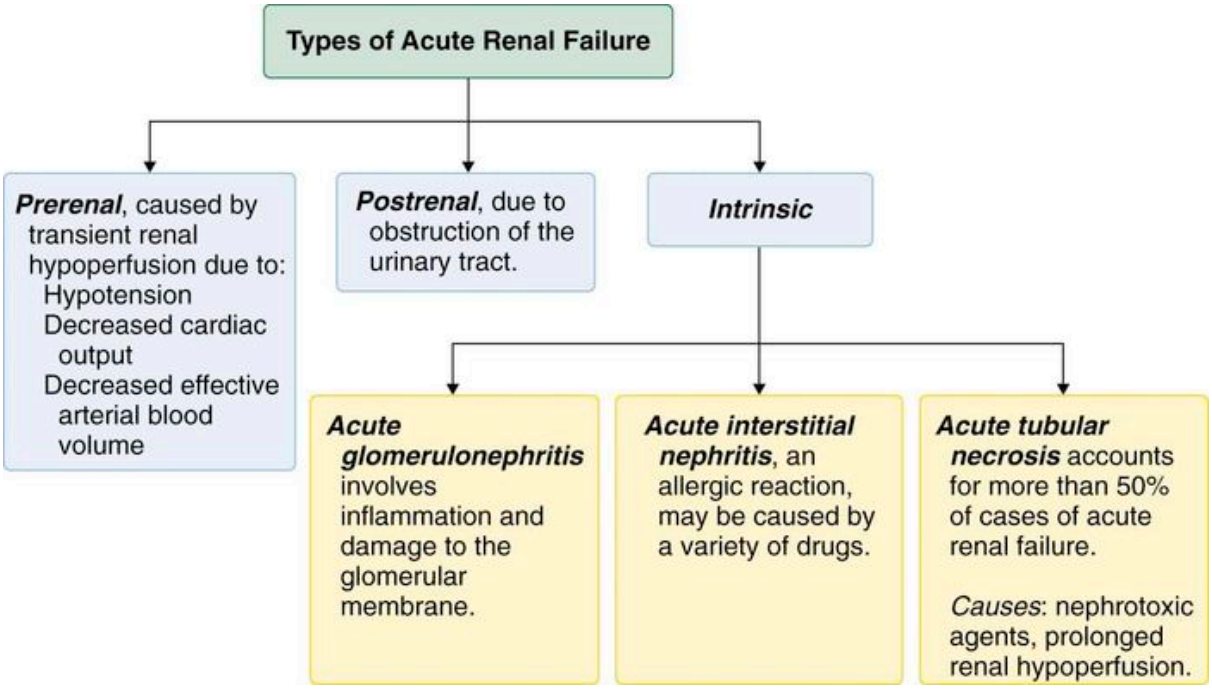
ETIOLOGY AND PATHOPHYSIOLOGY

AKI is broadly classified into 3 important categories ;

(i)Prerenal azotemia,

(ii) Intrinsic renal parenchymal disease

(iii) Post renal obstruction



Types of AKI.

PRE RENAL AZOTEMIA

Adequate renal perfusion is mandatory to maintain a normal GFR . Prerenal azotemia is due to decline in GFR caused by decrease in renal perfusion pressure with no damage to the renal parenchyma [3].Failure of the circulation in the body or exclusive failure of the intra renal circulation can show a large impact on the renal perfusion.

Decreased renal flow causes salt and water retention for restoring pressure and volume. when pressure and volume is decline the baro receptor reflexes which is located in carotid sinuses and aortic arch are gets activated .which leads to sympathetic nerve stimulations results in afferent arteriolar vasoconstriction and through B1 receptors Renin secretion [4].

Afferent arteriolar constriction leads to decline in intra glomerular pressure, which decreases the GFR in proportion. Angiotensin I is converted angiotensin II by renin; in turn release aldosterone .

Reduction in pressure or volume is a non osmotic stimulus for anti diuretic hormones from hypothalamus; which causes effect in collecting duct especially medullary collecting duct for water reabsorption. Through unidentified mechanism stimulation of sympathetic nervous system causes increased proximal tubular reabsorption of water and salt, also calcium, creatinine, uric

acid and BUN. The net result of above four mechanisms of water and salt retention is decline in output and urinary excretion sodium [<20 meq/l].

CAUSES FOR PRE-RENAL AZOTEMIA:

(i)Volume loss: [5]

Diarrhoea

vomiting

Heat exhaustion

Extreme sweat loss

Burns

Hemorrhage

(ii) Escape of fluid from circulation

Dengue

leptospirosis

Malaria & etc;.

(iii) Cardiac causes

Cardiac failure

Shock

Pericardial tamponade

Severe pulmonary hypertension

(iv)Interruption of blood flow to kidney

Renal artery embolism

Renal artery occlusion

(v)Renal loss

Diuretics

(vi) Decreased vascular resistance -peripheral vasodilatation

Sepsis

Vasodilator medications

Autonomic neuropathy

Anaphylaxis

INTRINSIC AKI

Sepsis, nephrotoxins, and ischemia are the most common causes of intrinsic AKI. Nephrotoxins are broadly separated as endogenous toxins and exogenous toxins. Many cases of prerenal azotemia progressed to tubular injury. Though classically named "ACUTE TUBULAR NECROSIS," renal biopsy confirmation of tubular necrosis is lacking in patients of ischemia and sepsis; Whereas, pathological processes such as apoptosis, inflammation, and altered perfusion in various regions of kidney may be relevant pathophysiologically. There are other causes of intrinsic AKI; which are less common .

Intrinsic kidney injury is anatomically conceptualised according to the site of parenchymal damage of kidney .(i) Glomeruli (ii)Tubulointerstitium, and

(iii)vessels.

ACUTE TUBULAR NECROSIS

Acute tubular necrosis (ATN) is a condition in which there is death of tubular epithelial cells that forms the tubules of the kidney. ATN is one among the cause of intrinsic acute kidney injury but it is more common and often seen in patients who has been hospitalized.

CAUSES OF ATN:

Surgery especially abdominal surgery or cardiovascular surgery.

Trauma to the kidney.

Extensive muscle injury or

Extreme physical exercise.

Toxic Substances to the kidneys any a times substances that are not toxic to the kidneys in a healthy individual may become toxic in a person who contracts diseases like CKD, diabetes, heart failure, or multiple myeloma.[6]

ACUTE GLOMERULONEPHRITIS

Acute Glomerulonephritis is a renal disease where there is an active inflammation of the Glomeruli . Both kidneys are composed of 2 million filtering screens known as glomeruli. Glomeruli screens and selectively remove nitrogenous waste products. In AGN the microvasculature in the kidneys become inflamed and damaged. The inflammatory process usually starts after injury (trauma) or infection; there by the protective immune system of the body fight against the infection; leads to scar tissue formation; and then

the process would be completed[7]. When the glomeruli is damaged it will not do proper filtering function.

AGN may also be caused by an abnormal immune response of the body.

CAUSES OF DIFFUSE GLOMERULO NEPHRITIS:

Lupus nephritis

Cryoglobulinemia

Good pasture's syndrome

Wegener's granulomatosis

Henoch- schonlein purpura

Peri arteritis nodosa

and other forms of vasculitis

Bacterial or viral infections

CAUSES OF AGN PRIMARILY AFFECTS KIDNEY:

IgA nephropathy

MPGN

Post infectious GN

ACUTE INTERSTITIAL NEPHRITIS

Acute interstitial nephritis is resulting from immune mediated inflamatory tubule interstitial injury, commonly initiated by medications like Antibiotics

such as Methicilin, Non steroidal anti inflammatory drugs like brufen,naproxen,infection & other causes.[8].Reaction to drugs causes 71%[9] to 92% [10]of AIN cases. Better to advice a patient to not to consume over counter medications.

CAUSES OF AIN:[11]

Medications:

NSAID

Penicillin

Methicillin

Sulphonamides

Furosemide

Thiazide diuretics

Allopurinol

Cyclosporin etc...,

Infection

Legionella

Leptospira

Cytomegalo virus

Mycobacterium tuberculosis

Ebstien-Barr virus

Polyoma virus

Ecoli etc...,

Autoimmune disorders

Kawasaki disease

Sjogrens syndrome

Systemic lupus erythematosis

Wegeners granulomatosis

Signs and symptoms of AKI:

Oliguria (though patients may have normal urine output)

Fluid retention – Pedal edema

Drowsiness

Fatigue

Shortness of breath

Confusion , Coma

Nausea ,Vomiting

Seizures

Hypertension

Easy bruising

Decreased appetite

Jaundice (in Liver disease)

POST RENAL ACUTE KIDNEY INJURY

Post acute renal kidney injury developed when there is an obstruction in the urinary tract below the level of kidneys which in turn directs wastes to concentrate in the kidneys .

Post renal acute kidney injury is not as common like intrinsic acute kidney injury.

An obstruction in the urinary tract may cause urine to accumulate in one or both kidneys. Over a period of time, this fluid which is build up will prevent the normal flow of urine out of the kidney.

CAUSES OF POST RENAL ACUTE KIDNEY INJURY

Stones - Ureteric calculous, Bladder stones

Prostate enlargement –BPH, Ca prostate

Blood clots in urethra and ureter

Cancer cervix

Renal cell carcinoma

Ca colon

Neurologic disorders - that impair bladder emptying- eg.., spinal cord injury, stroke , multiple sclerosis, and Parkinson's disease.

Relieving the obstruction is the mainstay of treatment . If the blockage is removed, the kidney gets recovered in 1 to 2 weeks if there is no other infection associated with.[12]

SIGNS AND SYMPTOMS OF POST RENAL AKI

Pain: severity- varies

location – Depends upon the type of obstruction. Pain felt in the lower back, lower abdomen, groin, genitalia

Difficult urination

Bladder Distension

Anasarca

Hypertension

Hematuria

Diagnosis of Post Renal AKI:

Postrenal AKI is diagnosed after an elaborate medical history and complete clinical examination with supra pubic distension of abdomen. Large amount of urine {2–3 litres} after catheterization of a bladder hold us to make the diagnosis. Following which the bladder swelling is resolved.

High risk of people who develops AKI

Diabetes mellitus Systemic hypertension

Heart failure

Multiple myeloma

Myeloproliferative disease

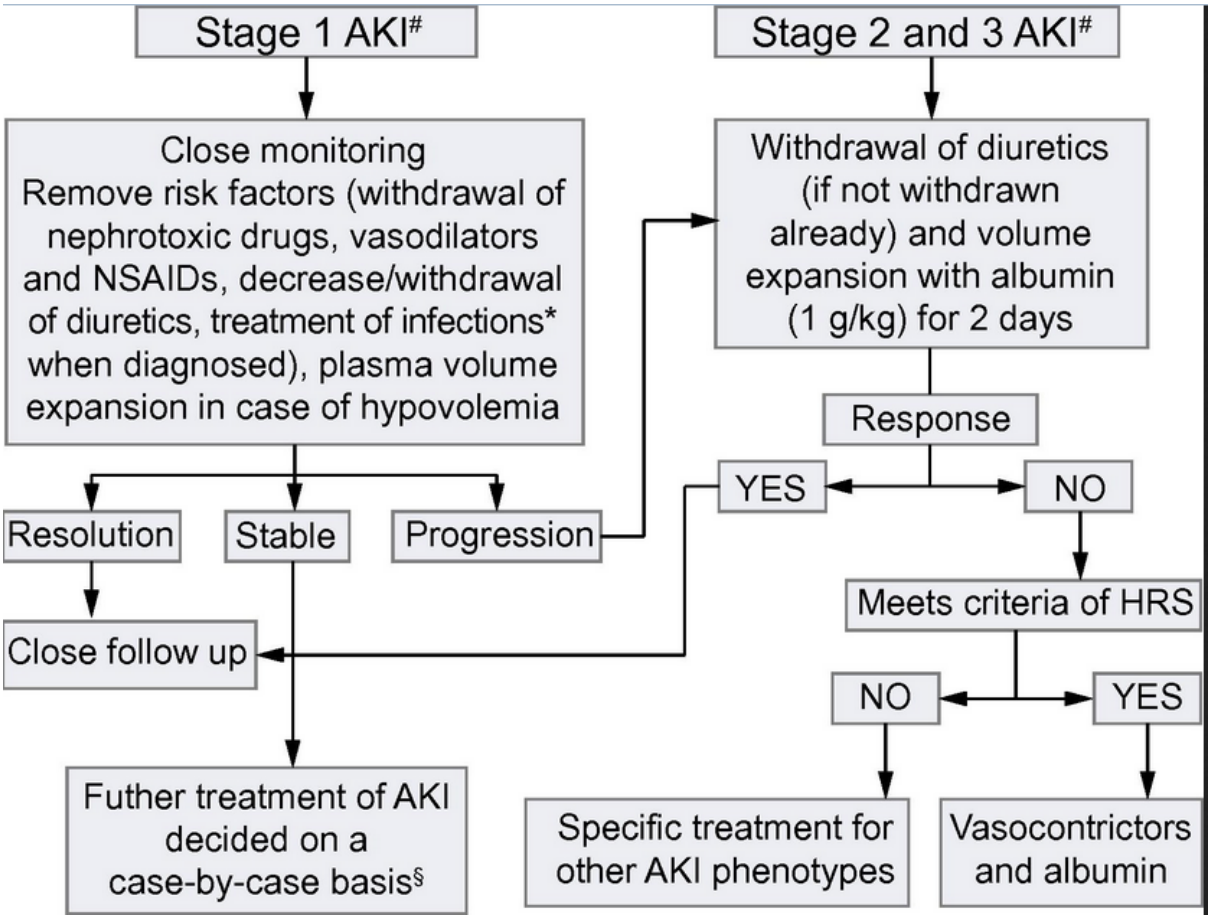
Autoimmune disease

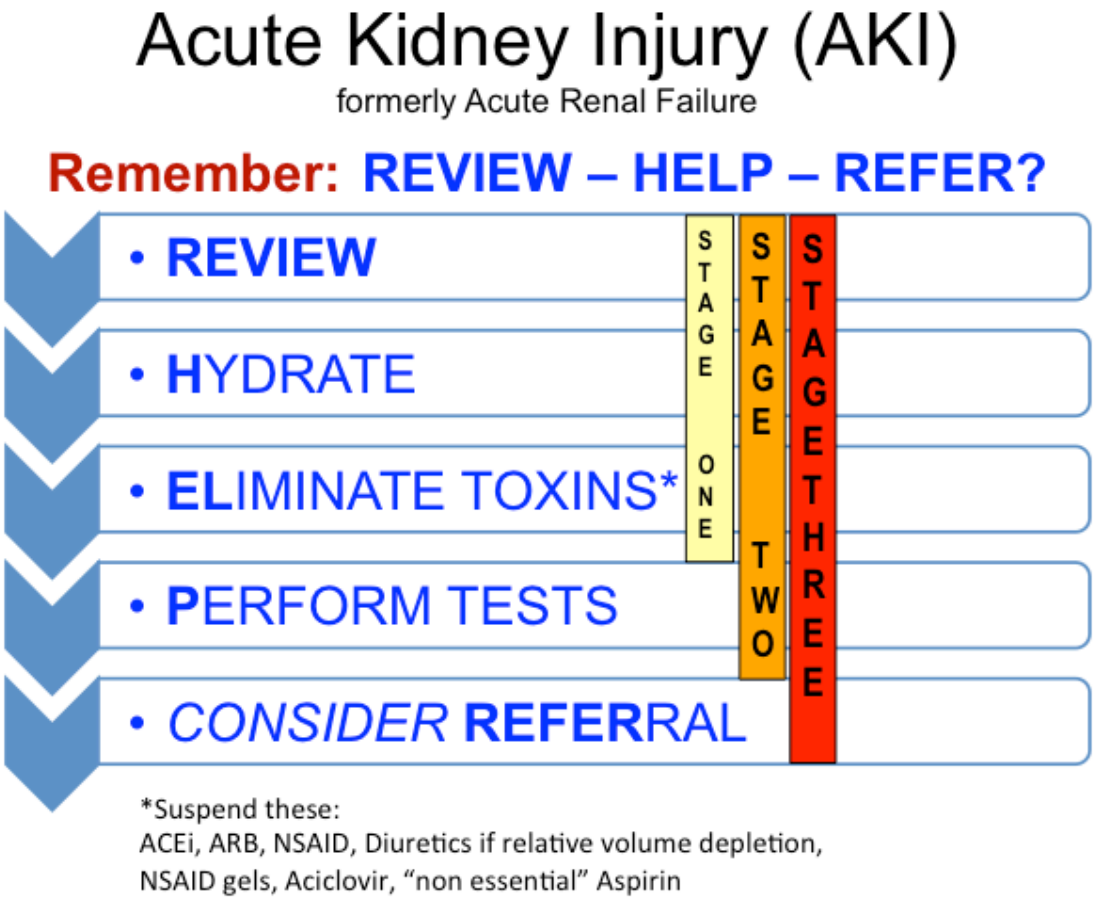
Connective tissue disorder and

Chronic infection[13]

AN APPROACH TO ACUTE KIDNEY INJURY

Management of AKI:





Management approach of AKI.

The acute kidney injury and chronic kidney disease both are not the different entity rather both are closely interconnected to each other. This fact is very important and useful to approach the patients while treating.[14]

As like other disease condition earlier the intervention better would be the out come of the disease.So biomarkers are more sensitive in acute kidney injury and is mandatory for the early intervention.[15]As of now FeNa, Serum creatinine, Spot urine and serum sodium are the available biomarkers to diagnose the established AKI.

BIOMARKERS

Since the creatinine is an imperfect biomarker for diagnosing AKI many biomarkers are under study to diagnose AKI early. The most promising biomarkers in diagnosis of AKI are interleukin -18, cystatin –C,Neutrophil gelatinase associated lipocalin – NGAL, kidney injury molecule -1.

NGAL-is expressed in renal tubules and it increases in urine before serum creatinine level increased in AKI. Urine and plasma NGAL predicts AKI in critically ill patients in ICU. It has both prognostic and diagnostic value.

KIM -1 – epithelial adhesion molecule ; present low level in normal kidney, increased in urine if the patient developed ATN.

Cystatin – C is produced by nucleated cells [16]. It is an early biomarker than serum creatinine. Accurate marker in contrast nephropathy.

TREATMENT

There are no therapies have emerged so far to expedite the recovery or else to attenuate the renal damage in AKI. So the treatment for AKI is supportive. If AKI is severe patient is treated with renal replacement therapy. Renal replacement therapies involved in treatment of AKI are Intermittent hemodialysis [IHD] and Chronic renal replacement therapy [CRRT].

The recent meta analysis of randomised trials for treatment of AKI with IHD and CRRT showed no difference in survival. Patients those who do not have chronic kidney disease as a premorbid condition will recover fast & come out of renal replacement therapy and become dialysis independent[17]. Also evidence says that patient who developed AKI are posing increased risk of subsequent chronic kidney disease.

KDIGO CLINICAL PRACTICE GUIDELINES FOR AKI

TREATMENT AND PREVENTION OF AKI:

- Guidelines suggest using isotonic crystalloids rather than colloids in the absence of hemorrhagic shock, as preliminary management for expansion of intravascular volume in patients with AKI.
- They have recommended the use of vasopressors in along with fluids in patients with vasomotor shock for patients at risk for AKI and patients with AKI.
- Guidelines suggests in ICU patients, insulin therapy with target plasma glucose of 110–149 mg/dl .
- Suggesting a total calorie intake of 20–30 kcal/kg/d in patients with AKI of any stage .
- No restriction of protein intake with the aim to prevent or delay initiation of Dialysis.
- Suggestion regarding administration of protein

  0.8–1.0 g/kg/d-- in non catabolic AKI, patients who does not require dialysis.

  1.0–1.5 g/kg/d --in patients with AKI on already with RRT

  1.7 g/kg/d-- in patients already on CRRT and inpatients with hyper catabolic condition.
- Guidelines recommend to not to use diuretics to prevent or treat AKI. Can be used in caution in volume overload status.

- It is not recommending to use renal-dose Dopamine ,Fenoldopam, Atrial natriuretic peptide and recombinant human (rh)IGF-1 to prevent or treat AKI.
- Single dose of Theophylline can be given in neonates with high risk of AKI suffered from perinatal asphyxia.
- It recommends not to use Aminoglycosides for infections unless there is no suitable, less nephrotoxic, antibiotics are available.
- Aminoglycosides are administered in cases with normal kidney function as a single dose daily rather than multiple-doses.
- Recommend aminoglycoside drug level monitoring when patient is treated with multiple doses for more than 24 hours.
- Suggested monitoring of aminoglycoside drug levels while treating with single-daily dosing for more than 48 hours.
- Suggested using topical applications of aminoglycoside rather than parenteral antibiotics, if necessary.
- Suggested lipid formulations of amphotericin B instead of conventional formulations.
- In systemic mycoses or parasitic infections, It recommends azole group of anti fungals agents or the echinocandins rather than amphotericin B, provided equal therapeutic efficacy is assumed.

GUIDELINES FOR CONTRAST INDUCED AKI

- In patients who develops derangement in renal function after administration of intravascular contrast agent, evaluate for CI-AKI
- Screen all the patients for pre-existing kidney function impairment to assess the risk for CI-AKI in whom the procedure intravascular administration of iodinated contrast medium is required.
- Alternative imaging methods can be considered in patients at increased risk for CI-AKI.
- Using possible lowest dose of contrast agent in patients at risk for CI-AKI.
- Recommended using either low-osmolar or iso-osmolar iodinated contrast medium, instead of high-osmolar iodinated contrast media in patients with increased risk of CI-AKI.
- Recommended intra venous volume expansion with 0.9% sodium chloride or sodium bicarbonate solutions, rather than without volume expansion, in patients at increased risk for CI-AKI.
- Suggested to not to use theophylline ,fenoldopam to prevent CI-AKI.

Suggested to not to intervene by prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for removal of contrast-media in patients at increased risk for CI-AKI.

TREATMENT OF AKI BY DIALYSIS INTERVENTION

- Initiate emergency RRT at the earliest when life-threatening changes in fluid, acid-base balance ,and electrolytes.
- Consideration about the broader clinical context, the positive clinical conditions that can be modified with RRT, and series of laboratory tests—instead of having single BUN and creatinine thresholds alone—when making the decision to start RRT.
- Discontinue RRT when it is not required, either because intrinsic kidney function has recovered to the point that it is fulfil the patient needs, or because RRT is not consistent with the goals of care.
- Use of diuretics is not suggested to enhance kidney function recovery, or to reduce the frequency or duration of RRT.
- In a patient requiring RRT with AKI, the decision to use anticoagulation for RRT based on the assessment of the patient's benefits and potential risks from anticoagulation
- Recommended use of anticoagulation while RRT in AKI when the patient does not have an increased bleeding tendency or impaired coagulation and is not receiving systemic anticoagulation already.

- For patients without bleeding risk or impaired coagulation and those who are not already receiving effective systemic anticoagulation, They suggest the following

  (i) In intermittent RRT, it is been recommended using unfractionated or low-molecular weight heparin, rather than HMW heparin anticoagulants. (ii)For anticoagulation in CRRT, suggested using regional citrate anticoagulation rather than heparin provided patients should not have contraindications for citrate.

  (iii) In patients who have contraindications for citrate, they suggested using un fractionated or low-molecular-weight heparin for anticoagulation during CRRT.

- Patients with increased bleeding risk who has not received anticoagulation, guidelines suggested the following for anticoagulation during RRT

  (i)Guidelines suggested to use citrate anticoagulation, rather than no anticoagulation, while patient is on CRRT with no contraindications for citrate.

  (ii) Guidelines suggested to avoid regional heparinization during RRT in a patient with high risk of bleeding.

  (iii) All heparin must be stopped in a patient with heparin-induced thrombocytopenia (HIT).

In such circumstances; guidelines recommend to use direct thrombin inhibitors like argatroban or Factor Xa inhibitors that is danaparoid or fondaparinux rather than no anticoagulation during RRT.

(iv) In a patient with HIT who does not possess severe hepatic failure, they suggest using argatroban rather than other thrombin inhibitors or Factor Xa inhibitors during RRT.

GUIDELINES ABOUT DIALYSIS

- Rather than a tunnelled catheter , Uncuffed non tunnelled dialysis catheter, is suggested to initiate RRT in patients with AKI .
- In patients with AKI, while choosing a vein for insertion of a dialysis catheter, consider the order of preferences

  (i)First choice: right jugular vein

  (ii) Second choice: femoral vein

  (iii) Third choice: left jugular vein

  (iv) Last choice: subclavian vein with dominant side preference.

- Recommended dialysis catheter insertion under ultrasound guidance.
- After placement and before first use of an internal jugular or subclavian dialysis catheter a chest radiograph recommended .

- Suggested to not to use topical antibiotics over the site of skin insertion of a non tunnelled dialysis catheter in patients with AKI at ICU requiring RRT.
- Suggested not to use antibiotic locks to prevent catheter-related infections for non tunnelled dialysis catheters in AKI.
- Suggested to use biocompatible membrane dialyzers for IHD and CRRT in patients with AKI.
- Use intermittent and continuous RRT as complementary therapies in AKI.
- Hemodynamically unstable patients they have suggested to use CRRT, rather than intermittent RRT.
- AKI patients with acute brain injury or patients with increased intracranial pressure or generalized brain edema guidelines suggested using CRRT, rather than intermittent RRT.
- Suggested using bicarbonate as a buffer in dialysate and replacement fluid for RRT in AKI, rather than using lactate.
- Recommended to use bicarbonate, not lactate, as a buffer in dialysate and replacement fluid for RRT in patients with circulatory shock and AKI.
- Suggested using bicarbonate, as a dialysate buffer and replacement fluid for RRT in patients with liver failure or lactic acidemia and AKI.
- Provide RRT to target to achieve the goals of electrolyte, solute, fluid balance and acid base balance that will meet the patient's needs.

OBJECTIVES:

1. **TO STUDY THE PATTERN OF ACUTE KIDNEY INJURY SEEN IN MEDICAL WARDS IN A TERTIARY CARE REFERRAL HOSPITAL**

2. **TO STUDY THE OUT COME OF ACUTE KIDNEY INJURY**

Materials & Methods

MATERIALS AND METHODS

STUDY DESIGN

Prospective observational study

STUDY PARTICIPANTS

Patients hospitalized in General Medical Wards Stanley Medical College

DURATION

January 2015 to August 2015.

PATIENT SELECTION

Any patients coming with elevated serum creatinine as per KDIGO guidelines and symptoms suggestive of acute kidney injury in the Medical wards.

EXCLUSION CRITERIA

Patients with Chronic Kidney Disease

SAMPLE SIZE

102 patients

METHODOLOGY

Patients admitted with elevated serum creatinine at Medical wards and intensive medical care ward as per KDIGO guidelines and symptoms suggestive of Acute kidney injury from JAN 2015 to AUG 2015 are included in this study. Patients will be subjected to symptom analysis , clinical examination appropriate laboratory investigations and imaging. Patient will be assessed for conservative medical management, Dialysis requirement and outcome of the disease . The final analysis will be made at the end of the study to achieve the aforementioned goals.

STATISTICS

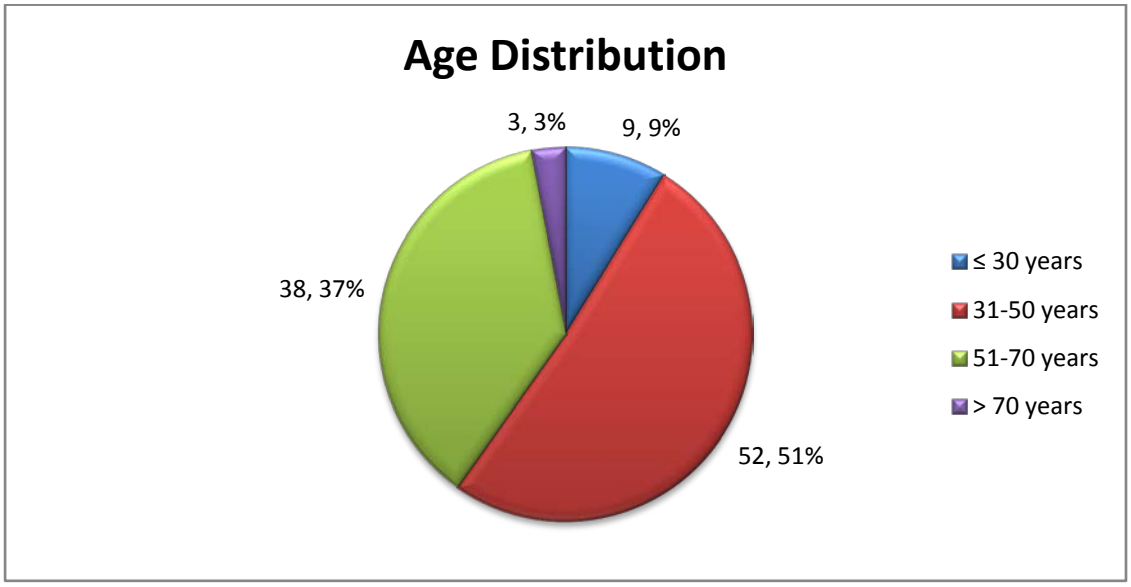
All the data were entered in a master excel sheet and analysed. The outcome of the study was analysed using multivariate logistic regression analysis to predict recovery in Acute Kidney Injury.

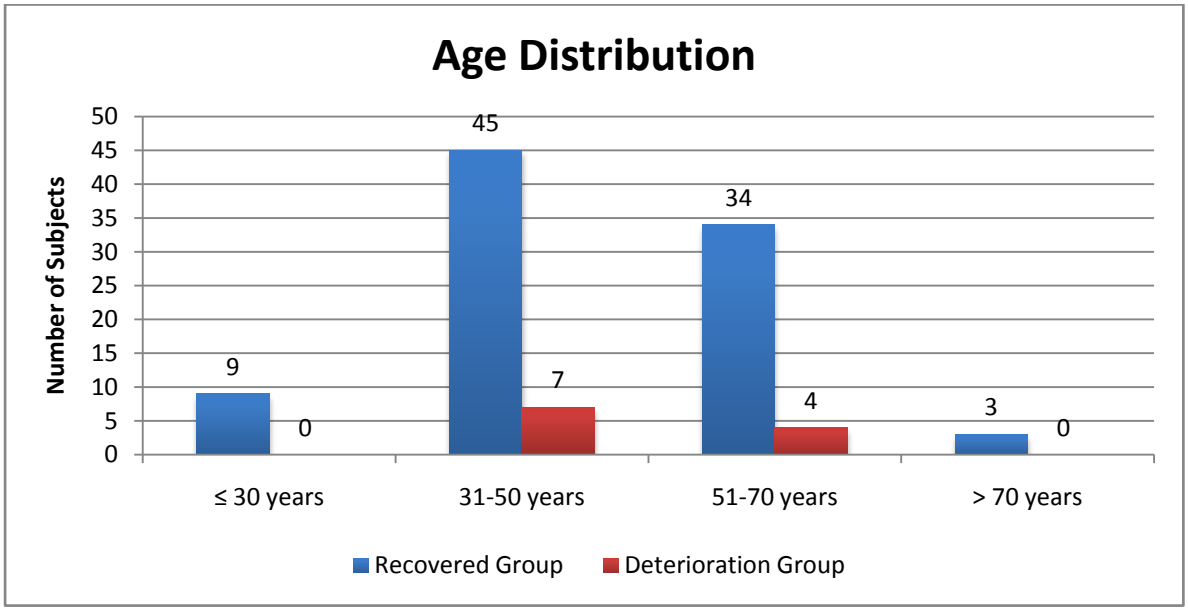
PREVALENCE

We conducted a 6 months prospective observational study of 102 patients those who were admitted at medical wards , using a relevant clinical database, to evaluate trends and outcomes of AKI seen in medical wards in a tertiary care referral centre Hospital

Results & Discussion

Age


Age Distribution
3, 3%
9, 9%
38, 37%
52, 51%
≤ 30 years
31-50 years
51-70 years
> 70 years

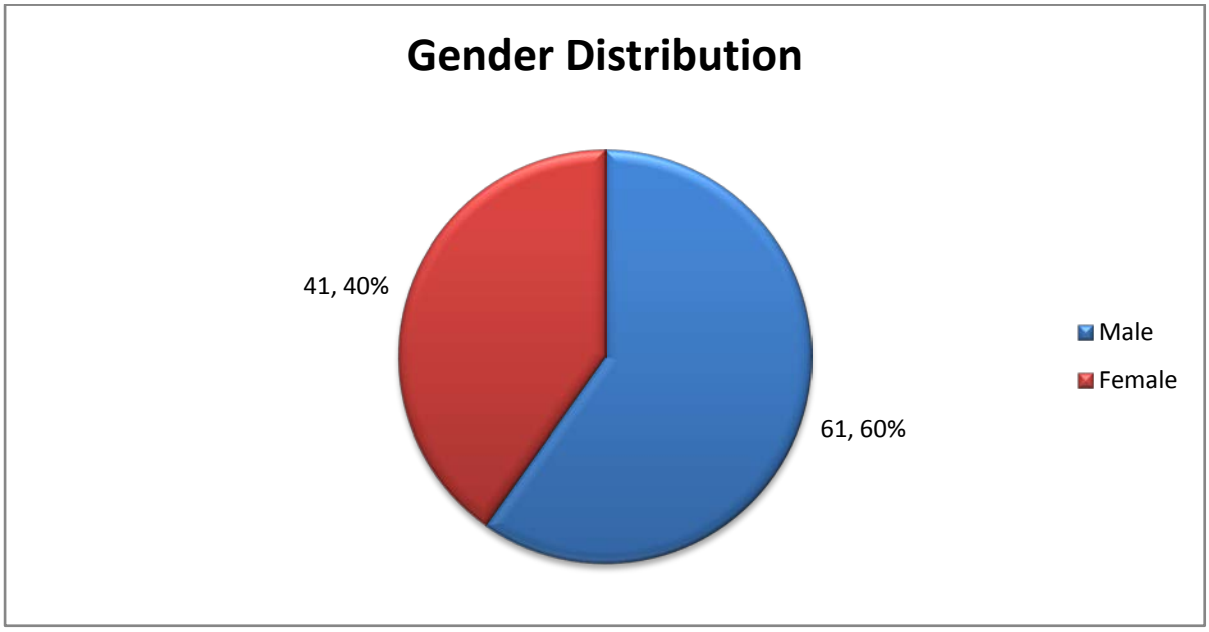

Age Distribution
Number of Subjects
50
45
40
35
30
25
20
15
10
5
0
9
0
45
7
34
4
3
0
≤ 30 years
31-50 years
51-70 years
> 70 years
Recovered Group
Deterioration Group

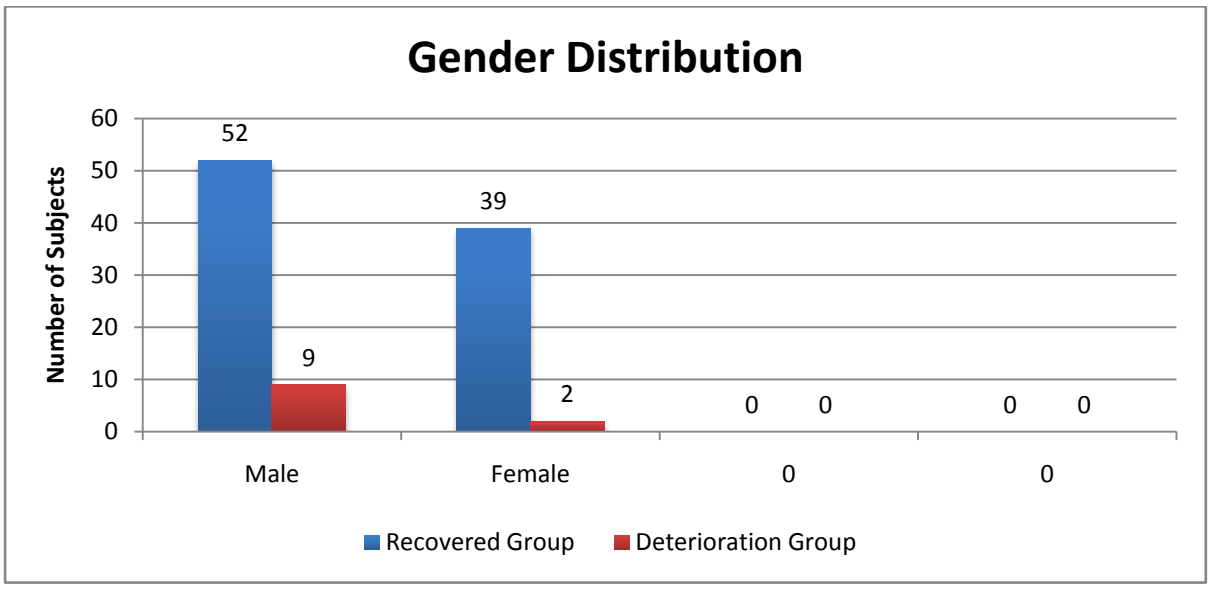
Age Distribution	Recovered Group	%	Deterioration Group	%
≤ 30 years	9	9.89	0	0.00
31-50 years	45	49.45	7	63.64
51-70 years	34	37.36	4	36.36
> 70 years	3	3.30	0	0.00
Total	91	100	11	100

Age Distribution	Recovered Group	Deterioration Group
N	91	11
Mean	47.59	49.09
SD	14.05	8.09
P value Unpaired t Test		0.6056

Majority of the recovered group patients belonged to the 31-50 years age class interval (n=45, 49.45%) with a mean age of 47.58 years. In the deterioration group patients, majority belonged to the same age class interval (n=7, 63.64%) with a mean age of 49.09 years. The association between the outcome groups and age distribution is considered to be not statistically significant since $p > 0.05$ as per unpaired t test.

Gender

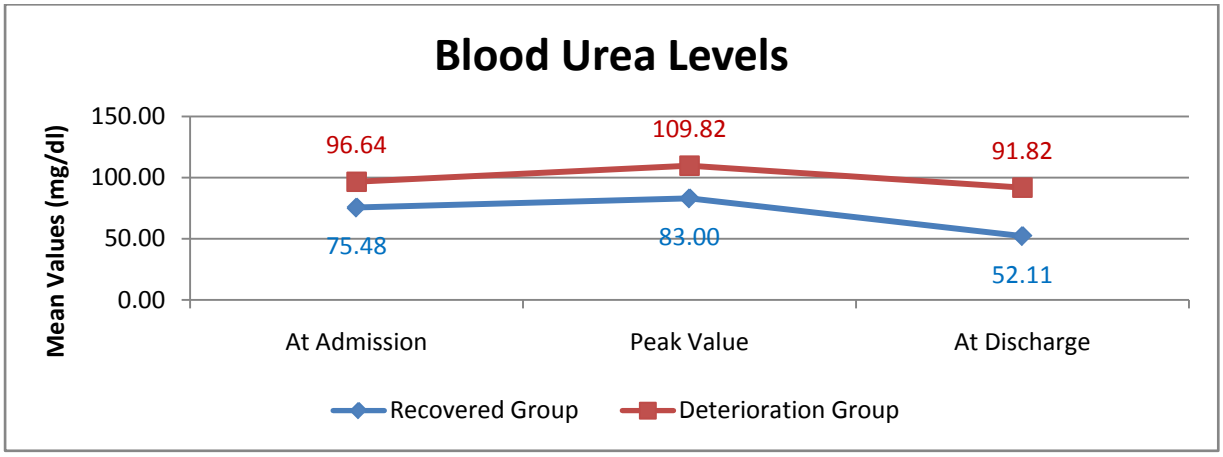

Gender Distribution
41, 40%
61, 60%
Male
Female


Gender Distribution
Number of Subjects
60
50
40
30
20
10
0
52
9
39
2
0
0
0
0
Male
Female
0
0
Recovered Group
Deterioration Group

Gender Distribution	Recovered Group	%	Deterioration Group	%
Male	52	57.14	9	81.82
Female	39	42.86	2	18.18
Total	91	100	11	100
P value Fishers Exact Test			0.1921	

Majority of the recovered group patients belonged to the male gender (n=52, 57.14%). In the deterioration group patients, majority belonged to the male gender (n=9, 81.82%). The association between the outcome groups and gender distribution is considered to be statistically not significant since $p > 0.05$ as per fishers exact test.

Blood Urea Levels

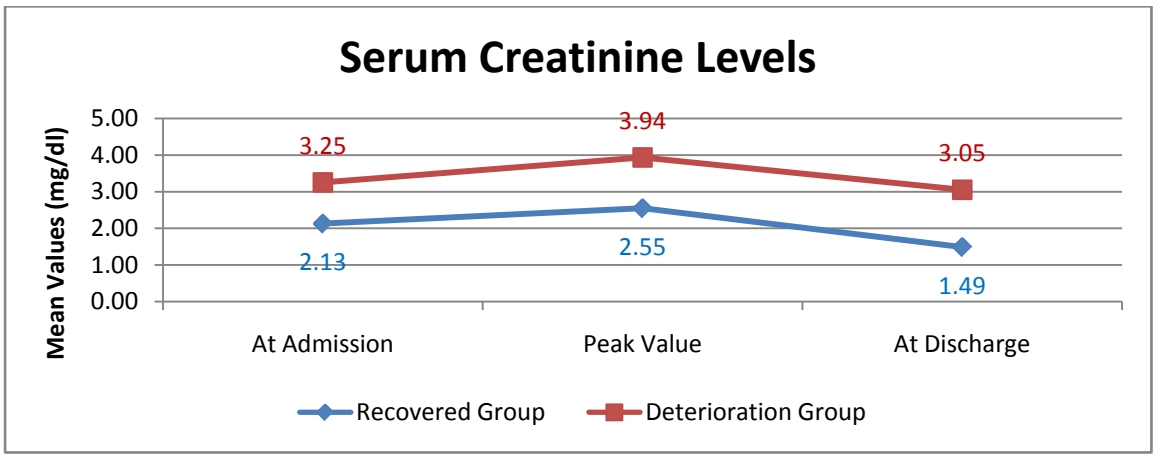


Blood Urea Levels		At Admission	Peak Value	At Discharge	Mean
Recovered Group	N	91	91	91	91
	Mean	75.48	83.00	52.11	70.20
	SD	31.44	35.34	26.28	28.22
Deterioration Group	N	11	11	11	11
	Mean	96.64	109.82	91.82	99.42
	SD	35.10	28.59	43.68	34.12
P value Unpaired t Test		0.0957	0.7572	0.6332	0.6181

The recovered group patients exhibited blood urea levels ranging from 75.48 mg/dl(at admission), 83.00 mg/dl (peak) and 52.11 mg/dl(at discharge) with a overall mean value of 70.20 mg/dl. In the deterioration group patients, majority exhibited blood urea levels ranging from

96.64mg/dl(at admission), 109.82 mg/dl (peak) and 91.82 mg/dl(at discharge) with a overall mean value of 99.42 mg/dl. The association between the outcome groups and blood urea levels distribution is considered to be not statistically significant since $p > 0.05$ as per unpaired t test.

Serum Creatinine Levels



Serum Creatinine Levels		At Admission	Peak Value	At Discharge	Mean
Recovered Group	N	91	91	91	91
	Mean	2.13	2.55	1.49	2.05
	SD	0.86	1.40	1.13	1.03
Deterioration Group	N	11	11	11	11
	Mean	3.25	3.94	3.05	3.42
	SD	1.65	1.43	1.98	1.57
P value Unpaired t Test		0.0489	0.0096	0.0262	0.0173

Results

In patients belonging to recovered group, the mean serum creatinine values ranged from 2.13 mg/dl(at admission), 2.55 mg/dl (peak) and 1.49 mg/dl(at discharge) with a overall mean value of 2.05 mg/dl. Similarly in deterioration group, the mean serum creatinine values ranged from

3.25 mg/dl (at admission), 3.94 mg/dl (peak) and 3.05 mg/dl (at discharge) with a overall mean value of 3.42 mg/dl. The decreased mean serum creatinine values in recovered group compared to the deterioration group is statistically significant as the p value is 0.0489, 0.0096, 0.0262 and 0.0173 at admission, peak, at discharge and overall mean respectively, as per unpaired t- test indicating a true difference among outcome groups.

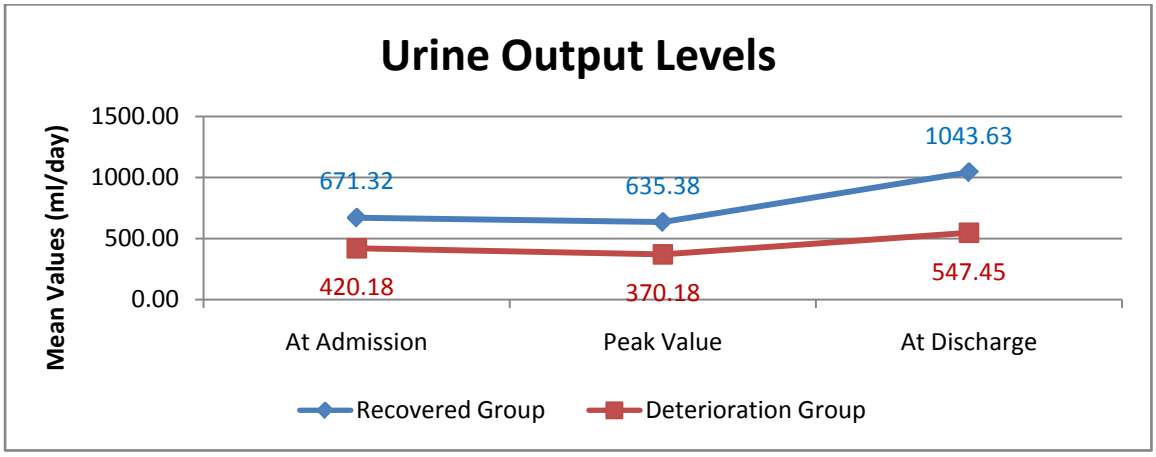
Discussion

The mean serum creatinine levels was meaningfully less in recovered group compared to the deterioration group by 1.36 mg/dl. This significant difference of 66 % decrease in mean serum creatinine levels in recovered group compared to the deterioration group is true and has not occurred by chance.

Conclusion

In this study we can safely conclude that mean serum creatinine was significantly and consistently lower in recovered group compared to the deterioration group. Hence we can infer that higher serum creatinine levels are associated with deteriorating and non recovery outcomes in AKI patients

Urine Output Levels



Urine Output Levels		At Admission	Peak Value	At Discharge	Mean
Recovered Group	N	91	91	91	91
	Mean	671.32	635.38	1043.63	783.44
	SD	242.90	283.84	359.93	275.00
Deterioration Group	N	11	11	11	11
	Mean	420.18	370.18	547.45	445.94
	SD	229.61	251.87	390.83	283.60
P value Unpaired t Test		0.0048	0.0062	0.0017	0.0027

Results

In patients belonging to recovered group, the mean urine output values ranged from 671 ml/day (at admission), 635 ml/day (peak) and 1043

ml/day(at discharge) with a overall mean value of 783 ml/day. Similarly in deterioration group, the mean urine output values ranged from 420 ml/day (at admission), 370 ml/day (peak) and 547 ml/day l(at discharge) with a overall mean value of 445 ml/day. The increased mean urine output values in recovered group compared to the deterioration group is statistically significant as the p value is 0.0048, 0.0062, 0.0017 and 0.0027 at admission, peak, at discharge and overall mean respectively, as per unpaired t- test indicating a true difference among outcome groups.

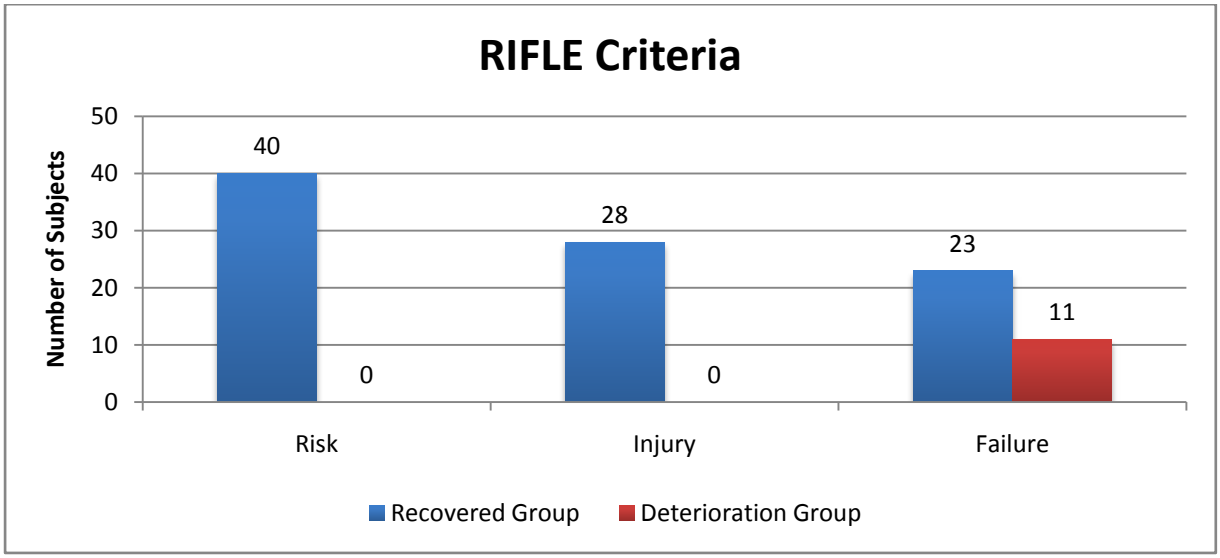
Discussion

The mean urine output levels was meaningfully more in recovered group compared to the deterioration group by 337 ml/day. This significant difference of 43 % decrease in mean urine output levels in recovered group compared to the deterioration group is true and has not occurred by chance.

Conclusion

In this study we can safely conclude that mean urine output was significantly and consistently higher in recovered group compared to the deterioration group. Hence we can infer that lower urine output levels are associated with deteriorating and non recovery outcomes in AKI patients

RIFLE Criteria



RIFLE Criteria	Recovered Group	%	Deterioration Group	%
Risk	40	43.96	0	0.00
Injury	28	30.77	0	0.00
Failure	23	25.27	11	100.00
Total	91	100	0	0.00
P value Fishers Exact Test			0.0388	

Results

In patients belonging to recovered group, the majority belonged to the risk category (n=40, 43.96%). Similarly in deterioration group, majority of the patients belonged to the failure category (n=11, 100%). The decreased incidence of failure RIFLE criteria in recovered group

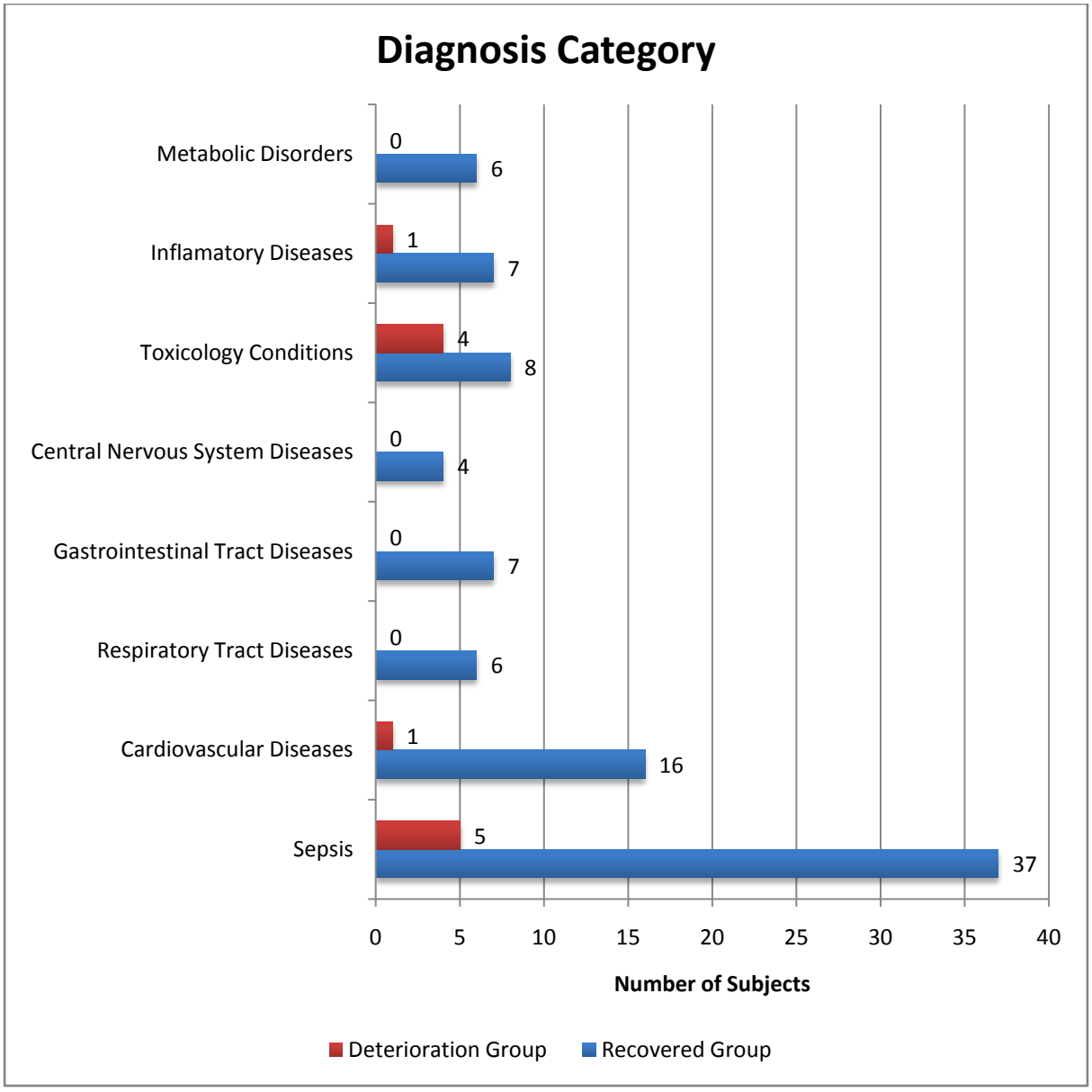
compared to the deterioration group is statistically significant as the p value is 0.0388 as per fishers exact test indicating a true difference among outcome groups.

Discussion

The incidence of failure RIFLE criteria was meaningfully less in recovered group compared to the deterioration group by 75 percentage points. This significant difference of 75 % decrease in failure RIFLE criteria in recovered group compared to the deterioration group is true and has not occurred by chance.

Conclusion

In this study we can safely conclude that the incidence of failure RIFLE criteria was significantly and consistently lower in recovered group compared to the deterioration group. Hence we can infer that higher incidence of failure RIFLE criteria are associated with deteriorating and non recovery outcomes in AKI patients


Diagnosis Category
Metabolic Disorders
Inflamatory Diseases
Toxicology Conditions
Central Nervous System Diseases
Gastrointestinal Tract Diseases
Respiratory Tract Diseases
Cardiovascular Diseases
Sepsis
0
6
1
7
4
8
0
4
0
7
0
6
1
16
5
37
0
5
10
15
20
25
30
35
40
Number of Subjects
Deterioration Group
Recovered Group

Diagnosis Category	Recovered Group	%	Deterioration Group	%	P value Fishers Exact Test
Sepsis	37	40.66	5	45.45	0.7523
Cardiovascular Diseases	16	17.58	1	9.09	0.3321
Respiratory Tract Diseases	6	6.59	0	0.00	0.5617
Gastrointestinal Tract Diseases	7	7.69	0	0.00	0.2065
Central Nervous System Diseases	4	4.40	0	0.00	0.9999
Toxicology Conditions	8	8.79	4	36.36	0.0030
Inflamatory Diseases	7	7.69	1	9.09	0.0956
Metabolic Disorders	6	6.59	0	0.00	0.8834
Total	91	100	11	100	

Results

In patients belonging to recovered group, the majority belonged to the sepsis diagnostic category (n=37, 40.66%). Similarly in deterioration group, majority of the patients belonged to the sepsis diagnostic category (n=5, 45.45%). The decreased incidence of toxicology diagnostic category in recovered group compared to the deterioration group is statistically significant as the p value is 0.0030 as per fishers exact test indicating a true difference among outcome groups.

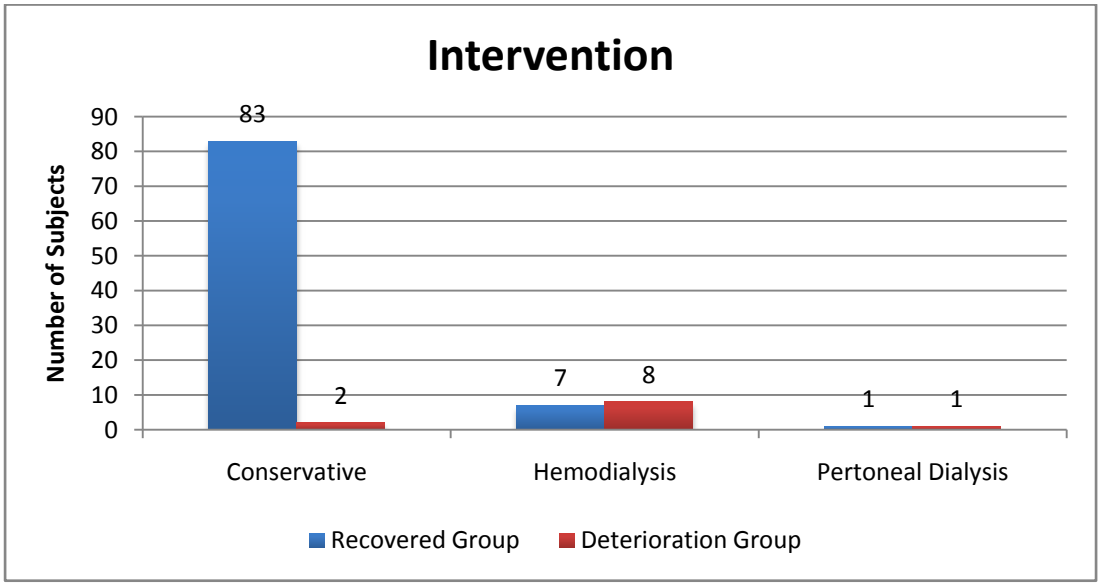
Discussion

The incidence of toxicological conditions as diagnosis was meaningfully less in recovered group compared to the deterioration group by 28 percentage points. This significant difference of 76 % decrease in toxicological conditions as diagnosis in recovered group compared to the deterioration group is true and has not occurred by chance.

Conclusion

In this study we can safely conclude that the incidence of toxicological conditions as diagnosis was significantly and consistently lower in recovered group compared to the deterioration group. Hence we can infer that higher incidence of toxicological conditions as diagnosis is associated with deteriorating and non recovery outcomes in AKI patients

Intervention



Intervention	Recovered Group	%	Deterioration Group	%
Conservative	83	91.21	2	18.18
Hemodialysis	7	7.69	8	72.73
Pertoneal Dialysis	1	1.10	1	9.09
Total	91	100	11	100

Multivariate logistic regression

Multivariate logistic regression model for statistically Significant Predictor of Deterioration in Health status

Independent Variables	Deterioration/Non Recovery		
	Odds Ratio	**95% Confidence Interval**	**P value**
Age > 40 years	1.76	0.80-53.35	0.0793
Male	1.5	0.69-15.51	0.1331
Peak Urea Values >100 mg/dl	1.24	0.61-8.78	1.236
Peak Creatinine Values >4.5 mg/dl	4.29	1.12-16.52	0.039
Peak Urine Output Values <400 ml/day	2.45	1.78-3.38	0.045
Peak Potassium Values >5 mmol/L	3.82	0.65-22.62	0.1393

The risk of deterioration/non recovery in patients with AKI having peak creatinine values > 4.5 mg/dl is 4.29 times significantly more than in patients with AKI having peak creatinine values < 4.5 mg/dl It is statistically significant with a p-value of 0.0039

The risk of deterioration/non recovery in patients with AKI having peak urine output values < 400 ml/day is 2.45 times significantly more than in patients with AKI having peak urine outputvalues > 400 ml/day. It is statistically significant with a p-value of 0.0450

DISCUSSION

Bagshaw et al.,[42] at 2007 had conducted a study , from 1996 January 1 to 2005 December 31 over all adult admissions at 20 Australian intensive care units (ICUs) for ≥ 24 hours with AKI. Trends in incidence and mortality for ICU admissions associated with early AKI were assessed. There were 91,254 patients got admitted at 20 study ICU s were studied.

The crude hospital mortality was significantly high for cases with AKI than those patients without ($P < 0.0001$). There was also a decrease in AKI crude hospital mortality ($P < 0.001$).After covariate adjustment, AKI still remained associated with a high mortality ($P < 0.001$).

Discussion: Over 1 decade, in a large cohort of critically sick cases admitted to Australian ICUs-20 centres, there had been a significant rise in the incidence of early AKI whereas the mortality associated with AKI has declined trend.

In this study , median age is of 64.1 (49 to 74.1) years, 60.6% were male.

In our study,majority of patients are between 31 to 50 years, 61.6% (similar with this study) of males. As per Bagshaw et al., study in incidence of AKI there were no significant changes stratified by age. In

the cumulative incidence stratified by sex there were no significant variation .

Conclusion of Bagshaw et al., study:

The incidence of AKI has increased in 1decade.

AKI at ICU admissions for metabolic cause and toxicology appears to have increased.

AKI possess an independent increased risk of death.

The associated mortality for patients with AKI remains high though declined over the past decade.

Hou *et al.*, in 1983 published prospective cohort studies of AKI [43]. The study focused on hospital-acquired disease and they excluded patients with AKI which is already established on admission. A total of 2216 patients were studied and followed up for the development of AKI.

Study found that the important causes of hospital-acquired AKI were due to decreased renal perfusion -42%, major surgery -18% , contrast nephropathy -12% , and Aminoglycoside use -7% . The crude in-hospital

mortality rate was 32%, change in serum creatinine was noted to be important to assess the degree of kidney injury.

In patients with an increase in serum creatinine of 0.5 to 0.9 mg/dl the in-hospital mortality was 3.8%, in patients with a serum creatinine of ≥4.0 mg/dl and in patients those who were not treated with renal replacement therapy the mortality increased progressively to 75% . This study established the association between oliguria and mortality in patients with AKI (52% *versus* 17% with and without oliguria, $P < 0.01$).

Discussion;

In our study similarly in deterioration group, the mean serum creatinine values are high compared to recovered group. The decreased mean serum creatinine values in recovered group compared to the deterioration group is statistically significant as the p value is 0.0489, 0.0096, 0.0262 and 0.0173 at admission, peak, at discharge and overall mean respectively, as per unpaired t- test indicating a true difference among outcome groups.

Liaño and Pascual., [44,45] , published a prospective study of all types of AKI episodes in 1991 in 13 tertiary-care hospitals in Spain.Their definition of AKI is as a sudden rise in serum creatinine of more than 2

mg/dl, they had excluded the patients with pre existing chronic kidney disease .

Of the 748 AKI patients acute tubular necrosis (ATN) was the most frequent cause (45%), includes diverse causes, like sepsis, surgery, renal hypoperfusion and nephrotoxin administration.

Next frequent cause was prerenal azotemia (21%), showed the rapid recovery of renal function following fluid resuscitation; acute on CKD (12.7%); and urinary tract obstruction (10%). Overall 45 % of crude in-hospital mortality rate was obtained and as high as 65.9% of mortality in patients requiring dialysis. Liano et al., had Compared ICU patients with non-ICU patients, those patients were admitted to the ICU were younger, more likely to die in-hospital (71.5% *vs* 31.5%), and they were more likely to have ATN from renal hypo perfusion, sepsis than nephro toxin administration.

Shusterman *et al., [46].,* Conducted a case-control study in patients with hospital-acquired AKI in 1981 . The incidence was 1.9% among patients on medical, gynaecological and surgical admissions. The 57 controls without AKI were matched to34 AKI cases.

This study found volume depletion, congestive heart failure , septic shock,, aminoglycoside use and intravenous contrast administration as risk factors for AKI. Also this study found a 10-fold increased odds of death and almost doubling time of the length of stay among the patients with AKI.

Dr. Alexandre Braga Libório, et al.,[47], Conducted a retrospective study by using data of Multiparameter Intelligent Monitoring in Intensive Care II project. Study period from 2001 to 2008. A total of 18,410 patients were participated in the study. A majority of the increased risk of mortality pertaining to AKI was attenuated by metabolic acidosis and cumulative fluid balance particularly in severe AKI. In particular, this study documented that RRT is associated with a better outcome in cases with AKI and its related complications.

Dr. Tariq Zulfiqar Ali, et al.,[48], Epidemiological study of acute kidney injury and acute-on-chronic renal failure . They had tested the hypothesis of Risk, Injury, Failure, Loss, and End-Stage Kidney (RIFLE) classification and predicts the outcomes. The incidences of AKI and ACRF were 1811 and 336 per million population, vice versa. Median age

for AKI was 76 yrs for and for ACRF 80.5 yrs. Sepsis was a causative factor in 47% of patients.

The RIFLE classification was very useful for predicting recovery of normal renal function ($P < 0.001$),requirement of renal replacement therapy ($P < 0.001$), in-hospital mortality ($P = 0.035$),length of hospital stay [excludes those who expired while admission ($P < 0.001$)]. The RIFLE criteria is useful to identify the patients at greater risk of adverse short-term outcomes.

Conclusion

CONCLUSIONS

1.Common causes of AKI in this study include,sepsis,cardiovascular diseases, drugs and poisons, and diarrhoeal disease in order of occurrence.

2.Among the patients who had AKI due to sepsis scrub typhus topped the list followed by leptospirosis and falciparum malaria.

3.Higher values of serum creatinine at admission and oliguria were the most significant factors that contributed to non recovery from acute kidney injury.

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Annexure

PROFORMA

- **NAME :** **SL. NO:**
- **AGE /SEX:**
- **OCCUPATION:**
- **ADDRESS WITH CONTACT NUMBER:**

- **DATE OF ADMISSION:**
- **DATE OF DISCHARGE:**

HISTORY

- H/o Decreased urine output:
- H/o Fluid retention- Anasarca
  Swelling legs
  Swelling ankles
- H/o Drug intake
- H/o Drowsiness
- H/o Shortness of breath
- H/o Loose stools
- H/o Vomiting
- H/o Fever
- H/o Fatigability

- H/o Confusion
- H/o Nausea
- H/o Chest pain
- H/o Hypertension
- H/o Diabetes
- H/o Seizures

Physical examination

- Built& nourishment
- Tachypnoeic
- Skin Hydration
- Anemia
- Icterus
- Bleeding
- Peripheral edema
- Anasarca
- Neck stiffness
- Asterixis
- Coma
- JVP
- Pulse: Bp: RR:

CVS -

RS -

P/A -

CNS -

INVESTIGATIONS

URINE ROUTINE ECG

CBC – TC CHEST X RAY

DC

ESR

HB

PLATELETS

BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

SR. CREATININE	ON ADMISSION	PEAK VALUE	ON DISCHARGE

GFR

SERUM ELECTROLYTES

Na	K	HCO3	Cl

LFT

TB	DB	SGOT	SGPT	SAP	TOTAL PROTEINS	ALBUMIN

USG ABDOMEN ECHO

OTHER PERTINENT INVESTIGATIONS

MONITORING OF URINE OUTPUT

DAY								
URINE								

OUTCOME

RECOVERY	
PARTIAL RECOVERY	
NO RECOVERY-HD DEPENDENT	
DEATH	

CAUSE OF DEATH

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC: "PATTERN OF ACUTE KIDNEY INJURY & ITS OUTCOME SEEN IN MEDICAL WARDS IN A TERTIARY CARE REFERRAL HOSPITAL"

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.

I understand that I will not get any payment for taking part in this study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer:
Signature of the investigator:

Signature/Thumb impression of the Volunteer
Dr.Jennie.S

Date:

Witnesses:
(Signature, Name & Address)

GOVT. STANLEY MEDICAL COLLEGE, CHENNAI – 600001

INFORMED CONSENT

PATTERN OF ACUTE KIDNEY INJURY & ITS OUTCOME SEEN IN MEDICAL WARDS IN A TERTIARY CARE REFERRAL HOSPITAL

ஆய்வாளர்:**மரு.ஜென்னி .,**

முதுநிலை பட்ட மேற்படிப்பு மாணவர்,

ப ெ மரு த்துவ பட்ட படிப்பு.

அரசு ஸ்டான்லி மருத்துவமனை.

<u>சுய ஒப்புதல் படிவம்</u>

பெயர்: வயது:

உள்ளிருப்புஎண்:

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களைக் கேட்கவும், அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது.நான் இவ்வாய்வில் தன்னிச்சையாகத் தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும், எந்த கட்டத்திலும், எந்த சட்டசிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை எனவும் அறிந்து கொண்டேன். என்னைப் பற்றிய தகவல்கள் இரகசியமாகப் பாதுகாக்கப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் .ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப எவ்விதமாகப் பயன் படுத்திக்கொள்ளவும், அதனை பிரசுரிக்கவும் முழு மனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன் ஆய்வாளருக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை தெரிவிப்பேன் என உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும், சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு

நோயளியின் கையொப்பம் ஆய்வாளர்கையொப்பம்

பெயர் (மரு.ஜென்னி .)

GOVT. STANLEY MEDICAL COLLEGE, CHENNAI – 600001

INFORMED CONSENT

PATTERN OF ACUTE KIDNEY INJURY ITS OUTCOME SEEN IN MEDICAL WARDS IN A TERTIARY CARE REFERRAL HOSPITAL

ஆய்வாளர்:**மரு**.ஜென்னி .,

முதுநிலை பட்ட மேற்படிப்பு மாணவர்,

ப ெ மர ் த ் வ பட ் ட படி ் ப ு.

அரசு ஸ்டான்லி மருத்துவமனை.

<u>பங்கேற்பாளரின் தகவல் படிவம்</u>

நீங்கள் இந்த ஆய்வில் பங்கேற்க அழைக்கப்படுகிறீர்கள். இந்த ஆய்வில் பங்கேற்கும் முன், இதன் நோக்கத்தையும், முறைகளையும், இதனால் ஏற்படும் பின் விளைவுகளையும் நீங்கள் அறிந்து கொள்ள ஆய்வாளர் அளிக்கும் தகவல்:

சி ற ீ ர க ந ே ் உள ் ள நோயாளிகள் இந்த ஆய்வில் சேர்த்துக் கொள்ளப்படுவார்கள். உங்கள் நோயின்வரலாறும், உங்களின் முழு உடல் பரிசோதனையும் தெளிவாகவும் விரிவாகவும் பதிவு செய்யப்படும். சி ற ீ ர க த ் த ி ல ் இர ் ந ் த வ ி ை ய ே ற ் ற ப ் பட வ ே ண ் ட ி ய உ ப ் ப ி ன ் அள ை இரத்தத்தில் பரிசோதனை செய்யப்படும்.

இந்த ஆய்வின் முடிவுகள் மருத்துவ காரணங்களுக்காகவும், மருத்துவ கல்விக்காகவும் பயன்படுத்தப்படும். இந்த ஆய்வு பற்றிய சந்தேகங்களுக்கு உரிய முறையில் விளக்கமளிக்கப்படும். தங்களைப் பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும்.

இந்த ஆய்வில் இருந்து எப்போது வேண்டுமானாலும் தாங்கள் எவ்வித முன்னறிவிப்பின்றியும், எவ்வித சட்ட சிக்கலும் இன்றி விலகிக் கொள்ளலாம்.

இந்த ஆய்வில் பங்கேற்குமாறு கேட்டுக் கொள்கிறேன்.

நன்றி,

ஆய்வாளர் கையொப்பம் நோயாளியின் கையொப்பம்

(மரு.ஜென்னி) (பெயர்:

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Patterns of acute Kidney injury and its outcome seen in medical wards in a teritiary care referal Hospital.

Principal Investigator : Dr.S. Jennie

Designation : PG in M D (General Medicine)

Department : Department of General Medicine
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.02.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

Master Chart

SL.	AGE	SEX	UREA			CREATININE			URINE OUTPUT			RIFLE	ELECTROLYTES		USG-KUB	DIAGNOSIS	SEPSIS	CVS	RS	GIT	CNS	TOXICOLOGY	INFLAMATORY	METABOLIC	ONCOLOGY	INTERVENTION	OUTCOME
NO.			ADMSN	PEAK	DIS	ADMSN	PEAK	DIS	ADMSN	PEAK	DIS	CRITERIA	Na	K													
1)	32	M	74	102	44	2.8	3.4	1.1	400	300	1100	FAILURE	132	4	NORMAL	CORROSIVE ACID POISONING						1				CONSERVATIVE	RECOVERY
2)	62	M	98	98	46	2.4	2.4	1.1	350	350	950	INJURY	119	2.5	NORMAL	ACUTE ENCEPHALOPATHY/SHT					1					CONSERVATIVE	RECOVERY
3)	28	M	112	112	42	2.2	2.2	1.1	400	350	1100	INJURY	134	4.2	NORMAL	HANGING/HIE/T2DM/SEIZURES						1				CONSERVATIVE	RECOVERY
4)	19	F	162	196	64	5.4	9.1	2.3	150	30	800	FAILURE	134	4	NORMAL	RAT KILLER POISONING						1				HD	RECOVERY
5)	34	M	98	138	90	1.2	5.8	1.8	950	300	1000	FAILURE	135	4.6	NORMAL	NH4 DICHROMATE POISONING						1				HD	HD DEPENDANT
6)	55	M	42	98	39	1.4	3.1	1.1	800	400	1200	FAILURE	132	3.6	NORMAL	SNAKE BITE -VIPER						1				CONSERVATIVE	RECOVERY
7)	34	M	62	96	43	2	3	1.1	550	400	900	FAILURE	129	3.8	NORMAL	ACUTEPANCREATITIS							1			CONSERVATIVE	RECOVERY
8)	55	F	58	90	38	1.1	2.8	1	900	390	1400	INJURY	136	4.4	NORMAL	LEPTOSPIROSIS/MODS	1									CONSERVATIVE	RECOVERY
9)	58	F	68	101	56	1.6	2.4	1.2	350	200	800	INJURY	139	3.4	NORMAL	CAD/ASMI/SHOCK		1								CONSERVATIVE	RECOVERY
10)	49	F	89	96	30	1.7	2.4	1	1000	1000	1600	INJURY	132	3.4	NORMAL	SCRUBTYPHUS/SEPTICSHOCK	1									CONSERVATIVE	RECOVERY
11)	19	M	186	198	159	3.8	5.8	5.6	100	50	50	FAILURE	130	3.2	NORMAL	CEREBRAL MALARIA/MODS/SEPTIC SHOCK	1									HD	EXPIRED
12)	52	M	100	100	48	3	3	1.4	900	900	1400	FAILURE	129	3.8	NORMAL	DENGUE/COAGULOPATHY/SETIC SHOCK	1									CONSERVATIVE	RECOVERY
13)	38	M	88	88	50	2.4	2.4	1.3	600	600	1500	INJURY	130	3.9	NORMAL	ALCOHOLIC/ACUTE PANCREATITIS							1			CONSERVATIVE	RECOVERY
14)	40	M	60	92	51	2.6	3.2	1.6	440	300	850	FAILURE	131	3	NORMAL	COMPLICATED MALARIA	1									CONSERVATIVE	RECOVERY
15)	37	M	74	92	42	2	2.8	1.1	500	320	850	INJURY	129	3.8	NORMAL	T2DM/ACUTE PANCREATITIS							1			CONSERVATIVE	RECOVERY
16)	36	F	66	89	43	1.4	2.2	1	800	650	1200	INJURY	136	3.8	NORMAL	SCRUBTYPHUS/SEPTICSHOCK	1									CONSERVATIVE	RECOVERY
17)	42	M	178	216	96	4.2	5.8	2	200	30	400	FAILURE	129	3.3	NORMAL	PT/ON ATT/ACUTE PANCREATITIS/?RIFAMPICIN	1									HD	HD DEPENDANT
18)	40	M	124	128	56	3	3	1.2	600	500	1000	INJURY	132	3.3	NORMAL	DENGUE HEMORRHAGIC FEVER	1									CONSERVATIVE	RECOVERY
19)	52	M	128	198	190	3	6.1	6.6	250	100	0	FAILURE	124	3.8	NORMAL	SNAKE BITE						1				HD	EXPIRED
20)	70	F	139	78	45	1.8	1.8	1.3	700	700	1100	RISK	134	3.6	NORMAL	CAD/AWMI/LV DYSFUNCTION		1								CONSERVATIVE	RECOVERY
21)	38	M	82	129	104	2.8	5.6	5.1	400	150	200	FAILURE	129	3.9	NORMAL	ACUTE PANCREATITIS/MODS							1			HD	HD DEPENDANT
22)	19	F	99	110	50	3	3.2	1	500	300	800	FAILURE	136	4.2	NORMAL	BPD/LITHIUM TOXICITY						1				HD	RECOVERY
23)	30	M	68	68	42	1.8	1.8	1	600	600	900	RISK	132	4.8	NORMAL	DCLD/HCV RELATED				1						CONSERVATIVE	RECOVERY
24)	50	F	91	90	56	3.2	3.2	1.6	550	550	790	FAILURE	137	4	NORMAL	DENGUE/COAGULOPATHY/SEPTIC SHOCK	1									CONSERVATIVE	RECOVERY
25)	40	F	60	60	45	2.2	2.2	1	450	450	1000	INJURY	136	4	NORMAL	T2DM/SHT/ADD	1									CONSERVATIVE	RECOVERY
26)	50	F	62.29	70	50	2.4	2.5	1.2	800	800	1100	INJURY	129	3.8	NORMAL	T2DM/SUBACUTE APPENDICITIS				1						CONSERVATIVE	RECOVERY
27)	45	M	68	76	51	2.1	2.4	1	450	400	900	INJURY	127	3	NORMAL	PT DEFAULTER/CAT II ATT/ADD			1							CONSERVATIVE	RECOVERY
28)	31	M	95	100	60	4	4.5	2	500	300	900	FAILURE	122	3.1	NORMAL	CVT/SSS THROMBOSIS/ACC HTN					1					HD	RECOVERY
29)	80	F	64	64	42	1.6	1.6	1	500	500	780	RISK	129	4	NORMAL	CAD/SEVERE LV DYSFUNCTION		1								CONSERVATIVE	RECOVERY
30)	60	M	74	74	47	2.5	2.5	2	1000	1000	1200	INJURY	136	3.8	NORMAL	DM/HTN/CAD/CCF		1								CONSERVATIVE	RECOVERY
31)	45	M	210	210	66	1.9	2.2	1.2	400	400	750	INJURY	132	4	NORMAL	DM/CELLULITIS/SEPSIS	1									HD	RECOVERY
32)	67	M	94	100	68	3	3.1	1.4	600	400	900	FAILURE	136	3.4	NORMAL	DM/UROSEPSIS/LRTI	1									CONSERVATIVE	RECOVERY
33)	45	F	80	80	62	2.3	2.3	1	900	800	1000	INJURY	138	3	NORMAL	CAD/CARDIOGENIC SHOCK		1								CONSERVATIVE	RECOVERY
34)	80	M	74	74	52	2.8	2.8	1	900	900	1300	INJURY	132	4	NORMAL	RIGHT ML CONSOLIDATION/SEIZURES			1							CONSERVATIVE	RECOVERY
35)	40	F	90	90	60	1.7	1.7	1.2	800	650	1100	RISK	128	4	NORMAL	HYPOTHYROID/NSAID ABUSE						1				CONSERVATIVE	RECOVERY
36)	54	M	99	110	53	1.9	1.9	1.2	700	700	1200	RISK	132	4	NORMAL	SCRUB TYPHUS	1									CONSERVATIVE	RECOVERY
37)	36	M	98	98	64	2.6	2.6	1.4	700	600	1000	INJURY	138	4.2	NORMAL	LEPTOSPIROSIS	1									CONSERVATIVE	RECOVERY
38)	62	F	100	112	98	3.4	3.4	2.4	200	150	500	FAILURE	129	3.9	NORMAL	DM/HTN/SEPSIS/MODS	1									CONSERVATIVE	EXPIRED
39)	42	M	84	84	60	1.8	1.8	1.1	750	750	1200	RISK	120	3	NORMAL	ACUTE GASTRO ENTERITIS	1									CONSERVATIVE	RECOVERY
40)	38	M	76	76	50	1.8	1.8	1	800	800	1100	RISK	136	4	NORMAL	DENGUE HEMORRHAGIC FEVER	1									CONSERVATIVE	RECOVERY
41)	58	F	52	52	41	1.5	1.5	1	900	900	1500	RISK	130	4	NORMAL	SHT/CVA/ANEMIA					1					CONSERVATIVE	RECOVERY
42)	43	F	56	56	40	1.4	1.4	1	1000	1000	1600	RISK	132	4	NORMAL	T2DM/AUTE LVF		1								CONSERVATIVE	RECOVERY
43)	57	F	51	51	38	1.7	1.7	1	750	750	1400	RISK	134	2.7	NORMAL	SHT/ADD/HYPOKALEMIA	1									CONSERVATIVE	RECOVERY
44)	84	F	186	186	66	3	3	1.4	500	500	950	FAILURE	137	5	NORMAL	T2DM/DKA/CAD								1		CONSERVATIVE	RECOVERY
45)	40	F	63	63	38	2.5	2.5	1.3	800	800	1300	INJURY	142	3	NORMAL	PT/LT LUNG COLLAPSE/SEPSIS			1							HD	RECOVERY
46)	55	M	80	88	46	2.1	2.3	1.3	600	600	1500	INJURY	145	4	NORMAL	CAD/CCF/CARDIOGENIC SHOCK		1								CONSERVATIVE	RECOVERY
47)	56	M	78	78	50	1.8	1.8	1.1	900	900	500	RISK	142	5	NORMAL	COMPLICATED MALARIA	1									CONSERVATIVE	RECOVERY
48)	42	M	60	60	45	2.2	2.2	1	400	400	1200	INJURY	136	4	NORMAL	T2DM/SHT/ADD	1									CONSERVATIVE	RECOVERY
49)	38	M	79	79	54	2.1	2.1	1.2	700	700	1200	INJURY	142	5	NORMAL	CAD/PUL.EDEMA/SHOCK		1								CONSERVATIVE	RECOVERY
50)	40	M	75	75	45	2.3	2.3	1.2	800	800	1200	INJURY	138	4.3	NORMAL	T2DM/DKA								1		CONSERVATIVE	RECOVERY
51)	65	F	66	66	36	1.4	1.4	0.9	1000	1000	1400	RISK	132	3.8	NORMAL	T2DM/HYPERGLYCEMIA/UTI								1		CONSERVATIVE	RECOVERY
52)	40	M	75	75	50	1.8	1.8	1	800	800	1100	RISK	136	4	NORMAL	LEPTOSPIROSIS	1									CONSERVATIVE	RECOVERY
53)	65	M	80	80	50	2	2	1.3	600	600	1200	INJURY	132	4	NORMAL	CVA/RT HEMIPARESIS /ADD				1						CONSERVATIVE	RECOVERY
54)	56	F	65	68	46	1.7	1.8	1.1	700	700	900	RISK	131	3.8	NORMAL	CAD/OLD IWMI		1								CONSERVATIVE	RECOVERY
55)	45	M	60	62	52	1.4	1.6	1	800	900	1200	RISK	129	5	NORMAL	CAD/T2DM/DKA								1		CONSERVATIVE	RECOVERY
56)	42	M	58	58	42	1.7	1.7	1.1	900	900	1400	RISK	132	4.2	NORMAL	ALCOHOLIC/ACUTE PANCREATITIS							1			CONSERVATIVE	RECOVERY
57)	60	F	50	52	40	1.4	1.4	1	1000	1000	1300	RISK	143	4.9	NORMAL	CAD/SEVERE LV DYSFUNCTION		1								CONSERVATIVE	RECOVERY
58)	29	M	54	54	38	1.3	1.3	1	800	800	1300	RISK	129	3.5	NORMAL	CEREBRAL MALARIA	1									CONSERVATIVE	RECOVERY
59)	41	F	60	64	42	1.6	1.7	1.1	900	1000	1400	RISK	142	3.7	NORMAL	SCRUBTYPHUS/T2DM	1									CONSERVATIVE	RECOVERY
60)	52	M	59	59	38	1.7	1.7	1.2	850	900	1000	RISK	145	3.8	NORMAL	T2DM/CELLULITIS/SEPSIS	1									CONSERVATIVE	RECOVERY
61)	50	M	68	70	44	1.6	1.7	1.1	600	700	1200	RISK	146	4.4	NORMAL	PLHA/CHRONIC DIARRHOEA	1									CONSERVATIVE	RECOVERY
62)	70	M	62	64	43	1.4	1.5	1	800	900	1300	RISK	129	3	NORMAL	CAD/ACUTE LVF/T2DM		1								CONSERVATIVE	RECOVERY
63)	48	M	59	63	46	1.6	1.8	1.1	600	600	900	RISK	136	4.1	NORMAL	DCLD/ETHANOL RELATED/PHT				1						CONSERVATIVE	RECOVERY
64)	39	M	64	65	46	1.3	1.4	1.1	800	800	1400	RISK	138	4	NORMAL	ALCOHOLIC/ACUTE PANCREATITIS							1			CONSERVATIVE	RECOVERY
65)	35	F	56	60	44	1.4	1.5	1.1	900	900	1400	RISK	141	3.7	NORMAL	AFI/SCRUBT TYTHUS	1									CONSERVATIVE	RECOVERY
66)	35	M	90	98	91	2.6	3	1.4	450	300	450	FAILURE	131	3	NORMAL	SNAKE BITE						1				HD	RECOVERY
67)	29	M	170	190	210	4.8	5.9	6	100	50	50	FAILURE	122	3	NORMAL	PARAQUAT POISONING						1				HD	EXPIRED
68)	45	F	56	58	42	1.4	1.4	1	900	900	1200	RISK	136	4	NORMAL	DM/HYPER TENSION/CAD/ACUTE LVF		1								CONSERVATIVE	RECOVERY
69)	63	F	60	62	46	1.3	1.4	1.1	800	800	1100	RISK	131	3.2	NORMAL	T2DM/DKA/CAD/CAD								1		CONSERVATIVE	RECOVERY
70)	58	M	60	60	45	1.6	1.5	1	700	900	1300	RISK	136	4.1	NORMAL	OLD PT/PT SEQULAE/ADD			1							CONSERVATIVE	RECOVERY
71)	44	M	58	58	42	1.7	1.6	1	700	900	1500	RISK	137	4.4	NORMAL	OLD PT/RML CONSOLIDATION			1							CONSERVATIVE	RECOVERY

72)	33	F	52	56	40	1.3	1.3	0.9	900	900	1500	RISK	136	4	NORMAL	DENGUE HEMORRHAGIC FEVER	1									CONSERVATIVE	RECOVERY
73)	63	M	70	74	42	2	2.1	1.2	600	800	1300	INJURY	132	4.2	PYLNPRTS	DM/PYELONEPHRITIS/SEPSIS	1									CONSERVATIVE	RECOVERY
74)	59	M	62	64	44	1.6	1.6	1.2	700	800	1100	RISK	116	1.9	NORMAL	T2DM/ADD/HYPOKALEMIA	1									CONSERVATIVE	RECOVERY
75)	43	F	112	122	96	3	3.8	2.6	300	300	400	FAILURE	118	2.8	NORMAL	TBM/ON ATT/SEPTIC SHOCK	1									PD	EXPIRED
76)	67	F	56	62	43	1.4	1.5	1	700	900	1300	RISK	136	4.7	NORMAL	CAD/IWMI/SHOCK		1								CONSERVATIVE	RECOVERY
77)	70	F	58	60	40	1.3	1.4	1	800	800	900	RISK	134	4	NORMAL	T2DM/CEREBRAL MALARIA	1									CONSERVATIVE	RECOVERY
78)	52	M	54	59	46	1.3	1.4	1	650	700	900	RISK	129	4	NORMAL	DCLD/PHT/HBV RELATED				1						CONSERVATIVE	RECOVERY
79)	56	F	66	66	46	2	2	1.2	700	800	1100	INJURY	131	3.4	NORMAL	LEPTOSPIROSIS/TRANSAMNITIS/SEPSIS	1									CONSERVATIVE	RECOVERY
80)	33	F	146	167	149	4	4.9	4	300	350	300	FAILURE	128	3.1	NORMAL	ACUTE FULMINANT HEPATIC FAILURE/SEPSIS	1									HD	EXPIRED
81)	58	M	90	90	50	1.7	1.7	1.2	600	600	900	RISK	134	4.2	NORMAL	CAD/AS/SEVERE LV DYSFUNCTION		1								CONSERVATIVE	RECOVERY
82)	59	M	60	66	44	1.6	1.7	1.1	1000	1000	1400	RISK	130	3.1	NORMAL	T2DM/DIABETIC ULCER/SEPSIS	1									CONSERVATIVE	RECOVERY
83)	39	F	56	56	41	1.4	1.4	1	1000	1000	1600	RISK	134	3.8	NORMAL	SCRUB TYPHUS/ARDS	1									CONSERVATIVE	RECOVERY
84)	39	M	160	213	200	4	5.9	5.6	200	100	100	FAILURE	128	2.9	NORMAL	PARAQUAT POISONING						1				HD	EXPIRED
85)	45	F	82	88	48	2	2.2	1.2	600	600	900	INJURY	132	2.8	NORMAL	LEPTOSPIROSIS/MODS	1									CONSERVATIVE	RECOVERY
86)	29	F	46	68	40	1	2	1	800	600	900	INJURY	132	4.2	NORMAL	CORROSIVE ACID POISONING						1				CONSERVATIVE	RECOVERY
87)	27	F	30	54	16	1.4	1.9	1	1000	1000	1200	RISK	138	3.2	NORMAL	ALCOHOLIC SEIZURES					1					CONSERVATIVE	RECOVERY
88)	45	F	91	91	43	2.1	2.2	1.2	650	600	950	INJURY	131	5.1	PYLNPRTS	T2DM/DKA/PYELONEPHRITIS	1									CONSERVATIVE	RECOVERY
89)	70	F	88	92	56	3	3.8	1	1200	1200	1300	FAILURE	137	3.8	NORMAL	CAD/SHT/ADD	1									CONSERVATIVE	RECOVERY
90)	60	F	43	54	30	0.9	1.5	1	900	800	1200	RISK	138	2.4	NORMAL	T2DM/NON HEALING DIABETIC FOOT	1									CONSERVATIVE	RECOVERY
91)	65	M	78	82	41	1.6	1.9	1.1	1000	1000	1300	RISK	132	4	NORMAL	CAD/UA/ISCHEMIC DCMP		1								CONSERVATIVE	RECOVERY
92)	48	F	89	96	52	2.1	3.6	1.4	500	500	900	FAILURE	139	3	NORMAL	CAD/OLD PT/PARA PNEUMONIC EFFUSION			1							CONSERVATIVE	RECOVERY
93)	53	M	77	107	49	2.4	3.1	1.6	700	500	900	FAILURE	142	5	NORMAL	T2DM/SHT/LT LEG CELLULITIS/VOL OVERLOAD	1									CONSERVATIVE	RECOVERY
94)	58	M	65	76	52	1.2	2	1.1	1000	1000	1200	INJURY	143	3.1	NORMAL	SHT/T2DM/DIABETIC FOOT/AGE	1									CONSERVATIVE	RECOVERY
95)	50	M	95	118	66	3.8	5.6	2.4	300	200	350	FAILURE	121	3	NORMAL	CAD/ACUTE LVF/ADD		1								PD	RECOVERY
96)	42	M	40	66	39	1.1	2.2	1	700	500	900	INJURY	134	5	NORMAL	ALCOHOLIC GASTRITIS/HEMATEMESIS				1						CONSERVATIVE	RECOVERY
97)	59	M	86	112	132	2.5	4.6	5.4	300	150	150	FAILURE	121	2.2	NORMAL	CAD/EXTENSIVE AWMI/SHOCK		1								CONSERVATIVE	EXPIRED
98)	39	M	50	61	43	1.2	1.8	1.1	650	500	800	RISK	129	3	NORMAL	DCLD/PHT/UGI BLEED				1						CONSERVATIVE	RECOVERY
99)	60	M	100	114	76	3.8	3.8	1.4	500	400	800	FAILURE	128	2.9	NORMAL	T2DM/ACUTE PANCREATITIS/ALCOHOLIC							1			HD	RECOVERY
100)	36	F	50	65	48	1.2	1.9	1	1000	900	1200	RISK	135	4.7	NORMAL	AFI/SCRUBT TYPHUS	1									CONSERVATIVE	RECOVERY
101)	50	M	69	98	76	2.5	2.9	1.6	1100	1000	1400	INJURY	124	4	NORMAL	SHT/ACUTE PANCREATITIS							1			CONSERVATIVE	RECOVERY
102)	45	M	44	54	38	1.2	1.8	0.9	800	1000	1200	RISK	129	3.1	NORMAL	CAD/T2DM/DKA								1		CONSERVATIVE	RECOVERY

HD= HEMODIALYSIS
PD=PERITONEAL DIALYSIS
PYLNPRTS= PYELONEPHRITIS
ADD=ACUTE DIARRHOEAL DISEASE
AGE=ACUTE GASTROENTERITIS
AFI=ACUTE FEBRILE ILLNESS
DCLD=DECOMPENSATED LIVER DISEASE
PHT=PORTAL HYPERTENTION
MODS= MULTI ORGAN DYSFUNCTION SYNDROME